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CONTENTS

The Pathological Aspects of Rheumatic Fever . . . 659
Clarence E. de la Chapelle

Treatment of Mental Disease in France at the End of the Eighteenth Century 670
C. P. Oberndorf

A Review of the Maternity Statistics of New York City for the Year 1939 679
Thomas J. Duffield

Library Notes:

Recent Accessions 717

Deaths of Fellows 719

AUTHORS ALONE ARE RESPONSIBLE FOR OPINIONS EXPRESSED IN THEIR CONTRIBUTIONS

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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

NOVEMBER 1940

THE PATHOLOGICAL ASPECTS OF RHEUMATIC FEVER*

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ALTHOUGH rheumatic fever is still considered a disease of undetermined origin, most authorities believe that it is infectious. Furthermore, many feel that it is closely related to streptococcal infections. There is also much evidence[1,2,3] to suggest that the disease is one of hypersensitiveness to some antigen, probably bacterial.

The widespread changes produced by rheumatic infection throughout the body are now considered to be intimately related even though the etiologic agent cannot be demonstrated in the involved tissues. The inflammatory lesions, although primarily of two types, namely exudative and proliferative, are not sharply limited and vary somewhat with the organ involved.

It is now recognized that the initial lesion of rheumatic fever originates in the supporting structures and the mesenchymal ground substance of the involved organ or tissues. The response to this injury includes edematous swelling of the collagen and elastic fibers with cen-

* From the Pathological Laboratories of Bellevue Hospital and the New York University College of Medicine. Delivered March 26, 1940, at The New York Academy of Medicine under the auspices of the New York Heart Association, in the Symposium on Rheumatic Fever.

tral necrosis accompanied by infiltration of a substance, probably fibrin, which is eosinophilic in staining affinity. The process has been labeled fibrinoid degeneration or swelling[3] (Klinge's "Frühinfiltrat").

Following the injury to the collagen, edema appears in the surrounding tissues, either widespread as seen in the inflamed joints, or local as in the subcutaneous nodules. Numerous polymorphonuclear leukocytes are also present throughout the involved area. These initial reactions are mainly exudative, irrespective of whether they occur in the joints or in the heart valves. Later, proliferating cells appear in these areas of altered collagen. They are rather large cells with basophilic cytoplasm like that of plasma cells, somewhat polygonal in shape and with irregular margins. Their nuclei are vesicular, deeply stained, and with prominent nucleoli. They may be multinucleated especially as seen in the myocardium as constituents of the Aschoff body.

McEwen's[4,5] studies of these cells as obtained at biopsy from subcutaneous nodules and with supravital staining, indicate that they are not phagocytic. He feels that they originate from primitive connective tissue (mesenchymal) cells. With ageing they become longer, produce more collagen and assume the characteristics of fibroblasts.

Joints: Since clinically one of the commonest manifestations of rheumatic fever is polyarthritis, we shall first consider the pathological changes in the joints. The inflamed joint presents both edema of the periarticular tissues and effusion into the cavity causing the swelling, heat and redness found at the bedside. The effusion consists of a gray or yellow turbid mucoid exudate, the cellular content of which usually contains innumerable polymorphonuclear leukocytes, both fresh and degenerating, and a few lymphocytes and monocytes. Bacteria have not been consistently demonstrated in the exudate.

Histologic study of the joints shows practically all of the characteristic rheumatic changes. There is early a diffuse lesion involving the synovia and its supporting tissue. Edema and wandering cells are among the initial changes. Swollen, palisaded, synovial cells may be seen as well as areas of focal necrosis. Fibrinous (fibrinoid) infiltration may involve the superficial layers of the synovia or penetrate into the joint capsule itself.

Later these involved areas are invaded by mononuclear cells. Since they are often arranged perivascularly and in spindle formation they closely resemble myocardial Aschoff bodies.

Less conspicuous, but demonstrable in active cases, are similar granulomata of various ages in the supporting periarticular tissues, the ligaments, aponeuroses, and even where tendons fuse with the periosteum. These arthritic and periarthritic alterations usually heal with no or little permanent damage to the joints.

Subcutaneous Nodules: These interesting manifestations of the rheumatic type of inflammation, encountered chiefly in children, are usually found in areas of physiological stress and strain; for example, in the vicinity of flexor-extensor joints. Grossly they differ considerably depending mainly on their age. At biopsy the younger are soft and jelly-like in consistency with ill-defined margins; the older nodules are firm, gray, fibrous-looking and well circumscribed.

Histologically, the findings also vary with the age of the nodules. In the early stages one finds foci of swollen collagen, some undergoing fragmentation with deposits of fibrinous material and edema fluid in which varying numbers of wandering cells may be sparsely scattered. Blood vessels may show swollen endothelium and other damage; there are microscopic hemorrhages. Surrounding them are collections of polymorphonuclear leukocytes, lymphocytes and typical granuloma cells.

Subsequently there is a gradual organization of the lesion with replacement by fibrous tissue, merging imperceptibly with the original supporting connective tissue. The granuloma cells become elongated, contain less cytoplasm and are separated by layers of newly formed collagen. Lymphocytes and plasma cells are scattered about. The tissue becomes less vascular, more intercellular collagen is deposited, many fibroblasts appear and, finally, only a fibrous scar remains.

Serous Cavities: Involvement of the serous membranes, other than the pericardium, in rheumatic fever is mainly of clinical interest because of the relative infrequency with which active lesions are found at necropsy. As in the case of the joints, the inflammatory process usually resolves completely and thus leaves no residual evidence of its previous existence. Therefore, they are apt to be found at necropsy only in those patients dying during an acute or subacute phase of rheumatic fever in which death is uncommon. However, their incidence would probably be greater at postmortem if they were more carefully sought in these acute cases.

Peritoneum: A focal peritonitis may be found in rheumatic fever. The lesion observed varies from that of fibrinous infiltration to typical

granulomata, particularly in the peritoneum covering the diaphragm and liver, less so in the perisplenic layer. The diaphragm itself is not infrequently the site of rheumatic lesions including fairly typical Aschoff nodules. The psoas muscle has also been described as showing involvement by the rheumatic process.

The peritoneum may also be involved indirectly by lesions produced in the gastrointestinal tract secondary to inflammation of blood vessels supplying this system.

Pleurae: The incidence of rheumatic pleuritis, as encountered clinically, has been given as from 3 to 10 per cent. No figures are available as to its incidence at postmortem but it is probably distinctly less, particularly if only the active lesions are included.

Focal or widespread areas of rheumatic inflammation may be seen in the pleura which resemble the pericardial lesion to be described later. At first a plastic type of exudate is met with but fluid soon makes its appearance. The effusion is turbid, and occasionally blood-tinged. Microscopically, considerable fibrin, desquamated cells, many polymorphonuclear leukocytes and only a few lymphocytes may be seen. McEwen[6] not only found a fairly high percentage of clasmatocytes and undifferentiated young connective tissue cells in pleural exudates, but also, and probably of more importance, a goodly proportion of desquamated mesothelial cells, singly and in groups. The fluid becomes absorbed leaving organizing exudates and causing the pleura to become thickened. Occasionally the fibrinous exudation between the parietal and visceral layers is replaced by fibrous tissue and permanent adhesions between the two layers thereby occur.

Lungs: The existence of a specific rheumatic pneumonia is still a subject for dispute, both clinically and pathologically. There are obvious reasons for this. In the interpretation of the pulmonary changes occurring in patients with rheumatic fever several difficulties arise including the presence of passive hyperemia of the lungs, atelectasis, and the co-existence of unrelated infective pneumonia. At the bedside these changes in the lungs in rheumatic fever may be readily missed since they are transient and rarely produce symptoms or any obvious change in the clinical picture. They consist mostly of migratory areas of focal pulmonary hemorrhage and consolidation and are seen only in the acute or subacute phases of rheumatic infection. Since death in these stages is not common and, since the lesions are usually transient, their

incidence at necropsy is low. Although many pathologists agree on the type of pathological lesions found, there is considerable disagreement as to their specificity.

In the early stages it is thought by some[3,7] that focal hemorrhages of non-specific inflammatory origin are the main lesions. Others[8,9,10] feel that there are more specific changes including a perivascular pneumonia which is considered to be closely related to perivascular lesions. There have been described areas of focal interstitial necrosis and perivascular hemorrhages extending into the alveoli and accompanied by leukocytes of all types. This is associated with a proliferation of interstitial and perivascular cells which is said to resemble that seen in the rheumatic granulomata already described. This reaction together with the vascular lesions and focal hemorrhages constitute what is said to be characteristic of a "rheumatic pneumonia." A final decision regarding the specificity of these changes is still to be rendered. In our experience in the Pathological Laboratories at Bellevue Hospital we have not encountered a satisfactory example of rheumatic pneumonia.

Vascular lesions: Widespread inflammatory changes in blood vessels of all types are now recognized as an important feature in rheumatic fever, Klotz,[11] VonGlahn and Pappenheimer,[12] Holsti,[13] and others have contributed freely to this subject. Rheumatic arteritis may involve the aorta, the pulmonary artery and its branches, the coronary arteries and also the small vessels in the viscera and in the periphery. However, when affecting the smaller arteries the process is recognized histologically only when other rheumatic lesions are present elsewhere, for in itself it is not specific for rheumatic fever. In medium-sized arteries it may resemble polyarteritis nodosa fairly closely, whereas in the aorta and pulmonary artery syphilis is the disease which simulates it.

Rheumatic aortitis is usually only detected histologically but occasionally the presence of yellowish, translucent intimal plaques in the root or the ascending portion of the aorta, or at the root of the pulmonary artery, may suggest its presence, particularly if other rheumatic lesions are also present.

In the acute or active phases the intimal changes consist of swelling of fibrils and proliferation of large mononuclear cells arranged in rows between elastic or collagen tissue bands. In the media focal collections of polymorphonuclear leukocytes may collect between degenerating muscle and elastic fibers. In the adventitia granuloma cells and poly-

morphonuclear leukocytes may be seen about the vasa vasorum and even discrete Aschoff nodules may form. The vasa vasorum may also present inflammatory changes. In the healed stage only dense avascular flame-shaped scars are seen in the vicinity of the vasa vasorum. The latter may show distinctly thickened walls and stenotic lumina.

Coronary arteritis is usually not recognized grossly in the acute phase. In the healed stage there may be thickening of the artery and stenosis of its lumen of sufficient degree to be seen with the naked eye.

Histologically, polymorphonuclear leukocytes and large cells with pyknotic nuclei infiltrate the swollen coats of the affected vessels. The endothelium is often raised by a subintimal deposit of fibrin. The media may undergo necrosis, and hemorrhage may separate the muscle fibers. The inflammatory cells may form perivascular collars in the adventitia. Thrombosis and aneurysm formation are extremely rare. When healing takes place there results an irregular growth of connective tissue in all three coats with a resultant nodular thickening of the vessels. At times it is difficult to differentiate the lesion from that of atherosclerosis.

In addition to the vessels just considered many types of rheumatic changes have been described[12,13] in the arteries in other portions of the peripheral vascular tree. The arteritis may involve one or all of the layers of the vessels. One of the simplest changes noted is mere swelling of the endothelium, in others it may be separated from the basement membrane by a structureless fibrin-like material. There may be fragmentation of the elastic tissue or the media may show areas of fibrinous infiltration. Occasionally the process extends to the adventitia with infiltration of polymorphonuclear leukocytes and red blood cells into the perivascular tissues. In this stage the appearance is very much like polyarteritis nodosa.[14]

Heart

Although I have kept the consideration of the heart and its lesions for the end of this presentation, it is by far the most important organ involved in rheumatic fever. It not only is responsible for the majority of deaths in this disease but is also the seat of most of the physical disability sustained by those affected.

Carditis is the chief visceral manifestation of rheumatic fever. Although in the correct sense of the term it signifies inflammation of the whole heart, through clinical usage it has become to mean involvement

of any portion of the organ. The myocardium and the valves are the structures involved in the majority of instances; more rarely the pericardium. The more severe the rheumatic inflammation the more likely are all three structures to be involved.

Heart valves and Endothelium: In the pathogenesis of valvular lesions in rheumatic fever emphasis is now placed on the primary involvement of the substance of the valve rather than on the implantation of the infectious agent on its surface. As shown by Gross,[15] Holsti[16] and others, there is a close relationship between the left auricle, the pericardial wedge adjacent to the annuli of the valves, the valve rings and the valve leaflets. This probably furnishes the correct interpretation of the initial process, namely, that the primary inflammation is in the valve substance (interstitium) with secondary changes on the surface epithelium leading to the formation of rheumatic vegetations. It has therefore been recommended[17] that the term "valvulitis" be employed to denote the endocardial changes occurring in the valves in rheumatic fever when accompanied by diffuse changes in the valve substance.

Others[18] have considered the first injury to be endothelial, recognizing that the endocardium is specially subject to physiological trauma, particularly at the line of closure where the cusps impinge. However, at this point it is well to mention that Swift,[19] Holsti[16] and others have described cases with marked interstitial valvular inflammation without verrucous lesions, usually in patients dying accidentally while in an early phase of the disease.

Small, firm, gray or pinkish gray verrucae along the line of closure of the leaflets, most often on the mitral and aortic valves, constitute the commonest lesion seen by the naked eye in active rheumatic valvulitis. Histologically the verrucae appear as pink-staining material protruding from the leaflet, denude of endothelium. The eosinophilic material usually extends into the swollen interstitium of the leaflet. Fibrin may be deposited on the superficial aspects of the verrucae. Where the verruca meets connective tissue, variable changes may be seen. Prominent are large mononuclear cells with basophilic cytoplasm, some elongated, and at times arranged in palisades at right angles to the surface of the valve. Polymorphonuclear leukocytes and eosinophiles are less prominent.

In addition, as previously mentioned, diffuse interstitial inflammation may be seen in the leaflet without verrucae or other changes being

visible grossly. Small vessels at the base of the leaflet are increased in number and protrude into the valve. The collagenous tissue is edematous and invaded by polymorphonuclear leukocytes and eosinophiles. Rarely Aschoff bodies may appear in the valve substance near the vessels. The valve rings usually present the same changes.

Since patients with rheumatic fever rarely die in the first episode, hearts presenting only acute or active lesions are seldom encountered at necropsy. The leaflets in rheumatic hearts usually exhibit combinations of fresh and healed lesions, suggesting the presence of a chronic active valvulitis or a recurrent inflammation in a previously damaged valve. Verrucae discernible on the edge of the deformed leaflets are evidence of an active process. In other instances signs of active inflammation may be only microscopic.

The main feature of the healing process in the valves is an increase of connective tissue elements with scarring. The leaflets are opaque, of limited mobility and fused at their junctions. The healed verrucae may leave a fibrous ridge along the line of closure. The semilunar cusps become shorter and the orifices enlarge. Where fusion of cusps occurs stenosis of the orifice develops. The chordae tendineae are thickened, especially at their insertion, and often they fuse together or with the papillary muscles. The valve leaflets become immobile, and stenosis of the orifices and insufficiency of the valves ensue. Capillaries and calcific deposits may be visible beneath the endothelium of the scarred valve. Ulceration may result when these calcific deposits lie close to the surface endothelium. Bland thrombi may be deposited, especially when the auriculo-ventricular valves are so involved.

In some instances, probably infrequent, the exudative phenomena may recede and leave little evidence beyond the presence of changes similar to those occurring in advancing age, or only localized areas of a healed valvulitis.

MacCallum[20] was the first to describe the mural endocarditis which occurs so frequently in the left auricle. It consists of grayish-yellow corrugations of the endocardium localized, as a rule, to the area just above the posterior mitral leaflet. It is of importance since it not infrequently acts as a nidus for the implantation of bacterial endocarditis.

In these cases the endocardium and subendocardium are infiltrated by eosinophiles, polymorphonuclear leukocytes and many distorted cells with crescentic or elongated nuclei arranged perpendicularly to

the endocardium. Beneath this reaction are present long bands of elastic tissue or swollen collagen, along which large granuloma cells ("Aschoff" cells) are arranged in palisades. Spindle-shaped Aschoff nodules are fairly frequent in the active stages of mural endocarditis.

As the process becomes chronic, endocardial reduplications develop with increased vascularity of the subendocardial layers and often hypertrophy of the auricular myocardium.

At this point it might be pertinent to say a word about mural thrombosis, particularly in view of the fact that active rheumatic inflammation may frequently be seen in the underlying auricular tissue.[21] Thrombus formation is most commonly confined to the left auricle or its appendage; less frequently in the right auricle and appendage, or bilaterally. Intraventricular thrombi are rarely encountered in rheumatic hearts.

Certain factors apparently favor the development of auricular thrombi. These include severe mitral stenosis together with congestive heart failure, auricular fibrillation and continued local inflammation.[21,22] Of these, the persistence of active inflammation in the auricular endocardium appears to be the main one.

Myocardium: Rheumatic myocarditis presents no gross features other than dilatation and occasionally hypertrophy. The characteristic lesion, only visible microscopically, is the submiliary nodule described by Aschoff in 1904 and which remains the most specific lesion of rheumatic fever. The Aschoff node forms about foci of swollen and fragmented collagen in the perivascular connective tissue and in the interstitial tissue between muscle bundles. Edema and exudation of polymorphonuclears are among the early changes. Later there appear the characteristic granulomata cells previously described, with abundant granular, basophilic cytoplasm, conspicuous nuclei and prominent hyperchromatic nucleoli. Some are large and multinucleated ("Aschoff" cells).

Aschoff nodules may be found anywhere in the myocardium but the sites of greatest frequency are (1) interventricular septum, (2) wall of left ventricle, (3) papillary muscles, and (4) in the wall of the left auricle.

During the healing stage the Aschoff body contains many lymphocytes and fibroblasts and finally a minute perivascular fibrous scar remains. Occasionally the acute inflammatory reaction may be wide-

spread. Leukocytes may collect near Aschoff nodules but may also be found throughout the interstitial tissue of the myocardium or at the margins of miliary foci of necrosis.

More common than uncomplicated acute rheumatic myocarditis is chronic active myocarditis, for even those patients dying in the course of an apparently first episode, may show healed or organizing lesions as well as fresh ones. Histologically the co-existence of healing or healed perivascular and interstitial miliary scars with Aschoff bodies in various stages of development is the striking feature of this type of rheumatic inflammation.

Pericardium: The early lesions of rheumatic pericarditis are usually found over the base of the heart, near the origin of the great vessels. There may be seen a layer of shaggy, yellowish fibrin covering the pericardial surfaces. When extensive it gives the appearance of the so-called bread and butter heart. Effusion is seldom excessive and rarely purulent. However, it commonly is sanguineous.

The histological recognition of the process is dependent largely on the finding of rheumatic lesions elsewhere in the heart. One reason for this is the fact that Aschoff bodies are only occasionally found in the pericardium. Few polymorphonuclear leukocytes are present in the exudate. The endothelium beneath the fibrinous exudate may be swollen but otherwise fairly well preserved. In the deeper portions there may be seen a proliferation of fibroblasts and endothelial sprouts and also new vessel formation. The latter may bleed, thereby causing the sanguineous character of the exudate.

Mild rheumatic pericarditis may heal without leaving any visible scar. Occasionally, localized areas of thickened pericardium resembling "milk patches" may be the only residue of previous rheumatic inflammation. In other cases the entire visceral pericardium is thickened although smooth and glistening. Fibrous adhesions between the pericardial layers are another end-result of organization. They may be localized or obliterate the entire pericardial sac. Adhesions may also bind the pericardium to neighboring structures, particularly to the mediastinum. However, it is of interest to note that rheumatic pericarditis rarely, if ever, produces a constrictive type of adherent pericardium.

The author is indebted to Doctor Irving Graef for his criticisms and suggestions in the preparation of the manuscript.

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TREATMENT OF MENTAL DISEASE IN FRANCE AT THE END OF THE EIGHTEENTH CENTURY*

C. P. OBERNDORF

It has been said of the historian that he writes in the present of events selected from the past with the hope that his interpretation and revaluation may serve as guidance for the future. Such a thought permits me to present a brief, long-forgotten, probably contemporaneously obscure and inconspicuous dissertation revealing the medical attitude toward mental disease in southern France just before the outbreak of the Revolution. Its arresting revelations may serve in some way to gauge our present position and to indicate how alert the psychiatrist of tomorrow must be if the psychiatric social gains of today are to be sustained.

Our glimpse into the past takes us back to Montpellier, an ancient city beautifully situated near the Mediterranean, with a school of medicine famous from the twelfth century. This school is probably most frequently remembered as the place where Rabelais, the physician, the priest and social reformer of the sixteenth century, studied medicine. Montpellier is introduced here because the thesis which forms the basis of this paper was published there in 1783 and such publication could have occurred only because this University, and also the neighboring University of Toulouse, had been centers of progressive thought for a long time. They numbered among their faculties and students many of the leaders in medicine, particularly several interested in mental disorders.[1]

As having come from Montpellier may be mentioned Jacques Dubois (1478-1555), the first French physician to write on mental disease, Andre du Laurens, who early in the seventeenth century studied melancholia, and the distinguished clinician Boissier de Sauvages (1706-1767), whose liberal influence continued at the University long after he had passed away. It was at Toulouse that Francois Bayle, at the request of the governmental parliament, undertook an investigation of

* Read before the Section of Historical and Cultural Medicine of The New York Academy of Medicine May 8, 1940.

the state of certain persons who pretended that they were demoniacally obsessed (1689). There too the noted Phillippe Pinel received his doctor's degree in 1775, before he sought the liberal academic atmosphere of Montpellier for postgraduate study.

We find that at Montpellier in 1783 one Franciscus Naudeau, who had just become an alumnus in medicine from the University of Montpellier, submitted for his doctoral dissertation a thesis entitled, "An Attempt to Treat Mental Disorders," "*Tentamen Therapeuticum de Animi Perturbationibus*,"[2] which the author adds, "By the Grace of God and under the auspices of Mother of God, in the presence of the most august physician of Montpellier, Louis, he will strive to defend." This I hope he succeeded in doing to the satisfaction of a formidable array of the faculty members before whom he was to present his thesis. I think that he would find little difficulty in maintaining many of his tenets before the American Board of Psychiatry.

As was still the custom of that time, the thesis is written in Latin with which my acquaintance over the years had become tenuous. But when I came across this little pamphlet in Geneva, the footnotes in French were so compelling that I thought the text might be worthy of translation. This was arranged upon my return through The New York Academy of Medicine and it has proved again how old is much which we consider new.

It is fair to assume that this little essay presents only current psychiatric thought of that time–a compilation of the ideas pervading psychiatry in that period. Little more could be expected of a recent graduate who had not yet fully completed the requirements for his medical degree.

The thesis of Naudeau is of particular interest because in its approach it antedates the dramatic innovations introduced for the care and treatment of the mentally deranged by Phillippe Pinel (1745-1826) at the Bicetre Asylum in Paris in 1793. The revolutionary tempest which swept through France in 1789 was due, in part, to the spirit of searching inquiry of Descartes and Voltaire and to the exaltation of the virtue of the natural man by the soul-racked Rousseau. It was an irrepressible surge against the unremitting oppression, injustice and cruelty meted out to the great mass of the population. The question arises whether Pinel merely epitomized in the field of psychiatry this sentiment growing in France or whether he actually originated the new

system and approach with which he is generally credited. (*Traité medico-philosophique sur l'alienation mentale*—1801.)

The young Dr. Naudeau begins his dissertation with quotations from what was probably considered the best in current literature on the subject of mental disease. When he has exhausted this source he begins to flounder and, as is so often true today of theses submitted for doctorate degrees, the study ends with an assembly of fragmentary references from poorly studied and at times irrelevant cases. Nevertheless his presentation reveals how intensely the moot problems which confront us today were intriguing psychiatrists in France one hundred and fifty years ago. The question of the psychological effects of drugs; the currently much-discussed question of the effects of shock in many psychopathic states; the influence of mental disease in the production of somatic symptoms; the value of the psychological approach in treatment; all these were under vigorous debate and investigation.

Naudeau gives a definition of insanity which is terse and valid, namely: "By diseases of the mind or mental perturbations we mean certain more vehement and disordered mental operations which depend neither on reason nor even on will power. Our own definition is borne out by various definitions thus far given by other authors." In confirmation of this remark he quotes the definition of the Greek Zeno, "perturbation of the mind means being opposed to reasoning rightly, while mental agitation is contrary to nature," and according to Galen, "it is a condition not governed by reason." Naudeau continues: "It is evident from countless case reports, and indeed those down through the ages, how easily mental diseases can produce various diseases, even serious and stubborn ones, while in certain other cases they cure them.

"Perturbations of the mind, although they mark a departure from sound reasoning, are often able by their very force to be of great advantage to the body. Physicists demonstrate that, although the tornadoes, hurricanes, lightning, and thunder raging through the air are often a calamity, they are useful beyond belief in re-purging the air of filth. In the same way, therefore, tempests raging in the human economy are not so constantly harmful that they do not sometimes benefit health to the greatest degree."

The author early introduces the effect of shock in causing body change, and mentions the type of shock, "(severe, sudden, unforeseen) which causes a disorder of the functions, or which changes the mode of

being of a person in health or in sickness. Violent shocks, especially when produced unexpectedly in the animal economy, may be followed by the most serious disorders, by atrocious diseases, and even by death. Fright may make a person suffer fatal apoplexy; sudden death from joy is possible; anger may cause bursting of the vessels and violent hemorrhages, apoplexy, epilepsy, convulsions and death."

The idea of shock pharmacologically induced through insulin, metrazol, or other drugs to induce a change in stubborn and stationary mental conditions of the schizophrenic group is distinctly a procedure of the present decade. However, this eighteenth century writer states: "On the other hand, shocks which are very moderate or are induced at the right time are often very useful, and they may occasion certain reactions, certain changes which help to restore a person's nature, to improve his functions."

He quotes at great length a case reported by Kaau Boerhaave, a nephew of Hermann Boerhaave, the famous Dutch clinician of the beginning of the eighteenth century, of an epidemic of a convulsive disorder in a children's ward in the Charity Hospital at Haarlem. The disease had attacked the children successively and the hospital authorities were unable to check it. Boerhaave, a man of impressive appearance and voice, was called in. He resorted to a method strongly reminiscent of the Paquelin cautery, a procedure once, and possibly still, used by some neurologists to produce a counterirritation. Witnessing its application during my intern days, the object of the cautery seemed to me to be the alleviation of the irritation of the physician because the patient's case had baffled the physician in his attempts to effect a cure by other means.

Boerhaave had hooks of a certain shape heated until red-hot in a stove of live coals and threatened to apply the red-hot iron to a particular spot on the arm of the first child who should have an attack of the convulsive disease. The shock of the threat of the cruel remedy had a pronounced deterrent effect on these children, but "the weakest of the children, excessively struck by this operation to which they were going to be subjected, died on the spot and all the others were cured."

Another case illustrates the effect of shock upon a girl in deep sorrow, "her trouble inclining to almost epileptic or hysterical outbursts with no remission or mitigation of trouble from remedies employed. When all hope had been given up a musket was accidentally discharged

at the patient's bedside. It worked so happy a change in the patient that she soon recovered definitely."

The protective value of anxiety, not too dissimilar to Freud's concept of anxiety being a warning signal against danger, is suggested by the following: "Anxiety (anxietas) sometimes eradicates, to the highest degree, the most inveterate mental defects, incurable types of insanity. It does so either because the mind (chastised and thoroughly frightened by the horrible torture from its very sad senses) is recovered, or because the mind, with a very serious struggle on every side, agitated and stricken in its sanctum sanctorum" (which we might designate the conscience or super-ego) "undergoes an internal change and thus gains a new quality."

The author mentions shame as a species of fear but makes the following distinction: "There is a difference between shame and fear: when one feels afraid, the surface of the body visibly blanches and is drawn; when one feels ashamed, one blushes all over or, even in the absence of blushing and transparency (a very rare occurrence), there is a sensation of heat which begins at the face and soon pervades the entire body. Nevertheless, there have been certain instances of mental perturbation resulting from shame and resulting in diseases." As proof of the influence of the effect of shame on a physical situation, he cites a case of an outstretched hand which a young girl was unable to retract. When she was suddenly exposed nude in the presence of several people the shock of the shame relieved the condition (subluxation?).

From shame the author proceeds, with an unpardonable failure to include the term "psychosomatic medicine," to the effect reported by various observers, of sudden joy upon the relief of many conditions. These conditions include gout, indefinite fevers, arthritis, catalepsy, chlorosis and constriction of the pylorus. In connection with joy, for all his youth, Naudeau slyly remarks: "Nature herself long ago taught the oldest races of the East that cold and withered old men grow warm, and are miraculously revived, wheresoever they recline with a beauteous maiden. Any person who deems that all the healing virtues of this practice reside exclusively in the effluvia and welcome heat given off by youthful bodies, and who gives no credit to the sparks of love elicited conjointly (no matter how evanescent such sparks may be), will indeed find it difficult to explain why the fair sex was selected to do the warming."

He also discusses in considerable detail the value of music and the influence of hope in the treatment of depressions: "if hope is the result of faith and fear, then, I say it can aid the infirm." It is the "force which charlatans know so well how to bring into play, on which they base their reputation and their fortune, and to which we must attribute the cures worked by certain charlatans. The confidence of their patients, which results from the ignorance and from the blind credulity of the latter, determines the success of the quacks' remedies."

Naudeau's reflections on the use of drugs in mental disorder would receive the endorsement of the leaders of the profession today. "We must not seek to employ a host of drugs to repair the bodily disorders which may be wrought by mental disorders. Moral support is the only remedy to be tried, and the only one that offers hope of curing diseases referable to behavior. While Nature fails to function properly, while disorder affects her movements and functions, she cannot efficaciously direct the impression and the effects of remedies, which then only in vain cause loss of strength, increase the trouble, and aggravate the disease. This does not mean that care of the body is to be abandoned altogether; the body always presents sympathetic reactions, and often suffers even seriously when the mind is affected." Indeed, he quotes, psychosomatically speaking, from Sterne's Tristram Shandy: "The body and the mind are as a jerkin and its lining–rumple the one, you rumple the other." "But it does mean that the simplest and mildest remedies are the ones best suited to meet this requirement, which is merely secondary. The drastic means are to be directed against the mind, whose disease forms the principal and dominating indication."

Perhaps the most thought-provoking aspect of the thesis is that, aside from therapeutic shock, a psychological approach is the only one which Naudeau has acquired from his preceptors and his reading. He summarizes this in the following interesting technical suggestions: "It requires above all that the physician be fully acquainted with all the resources which he may find necessary, and possess the sublime and difficult art of using these resources. He must be able to make good use of eloquence which is gentle, touching, persuasive or cutting, strong, pressing, and irresistible. He must be able to inspire, to evoke at will the most contrary actions, feelings, and passions; love or hate, joy or sorrow, fear or hope, sweetness of disposition or anger, boldness or shame, etc. He must know how to subjugate the patient's mind cleverly and

to deserve the full confidence of the patient, for this ability works cures in the hands of certain physicians, cures sought by others in vain. On some occasions he must know enough to yield to the caprices of his patient, in order to lure him more easily into the traps cleverly set. The physician must, through the dominating affection and defect, seek to discover what things might impress the patient's mind and divert his attention in the opposite direction. Hence the physician must know how to employ all kinds of distractions to the right degree, amusements in the country and in society, music, dancing, the theatre, games, etc." This last sentence, phrased slightly differently and embellished by studied photographs, might well have been taken from the latest seductive prospectus of a contemporary progressive sanitarium for mental disease.

In the entire thesis of the young doctor we find no allusion to the dialectics of classification which interested and occupied the few French psychiatrists before Pinel.[1*] Nor is there any reference to commitments or to the cruel asylum practices, the punishments, the dungeons, the chains, the lack of understanding or even of a desire to understand, such as Pinel found at the Paris institutions. Apparently the beginnings of modern psychiatric thought had already taken firm hold before Pinel's time and Pinel's great distinction rests in the fact that his conviction of the correctness of this attitude was so complete that he had the courage to insist on its introduction into closed institutions which, then as now, were apathetic, if not hostile, to the reception of changes in methods.

Probably more important even than his mere advocacy of humane ideals was Pinel's determination that they could and should be practiced and not be represented by regulations behind which antiquated attitudes and procedures survived. Too often a humane theory without the spark of forceful sympathy loses its essence. This appears to have been the fate, partially, of Pinel's reforms at Salpêtrière. For, when Dr. Pliny Earle,[3] one of the pioneers in American psychiatry, visited that hospital in 1838, he was grieved and incensed to find Pinel's successor, Leuret, using the stream douche as an instrument of moral discipline, coercion and punishment. Yet, at that time, the distinguished clinician, Esquirol,

* In this bulky work detailing the contributions of each psychiatrist in biographical form there are evidences of the growing indignation against the maltreatment of the insane before Pinel, e.g., Lorry (1726-1783) declared that to treat maniacs by beating or confinement in irons intensified their excitement (volume I, p. 73) and Daquin published a book entitled "La Philosophie de la Folie, où l'on prouve que cette maladie doit plutôt etre traitée par les secours moreaux, que par les secours physiques," Chambery, 1791. Naudeau, the author, is not mentioned in Semelaigne's work and I have been unable to ascertain whether he continued his interests in psychiatry.

a disciple of Pinel, had transferred to Charenton Hospital the delicate task of studying and understanding human conflict and perpetuating psychiatric ideals current before Pinel's day.

I do not wish to leave the impression that in this rediscovery of a precedent approach to mental disease which has points in common with our own, I merely reaffirm a very old but only partially true observation from Ecclesiastes 1:10—"Nothing under the sun is new; neither is any man able to say, Behold this is new. For it hath already gone before in the ages that were before us." Many thoughts seemingly new to each generation have in some reminiscent form been known to antecedent centuries. Progress, however, does not subsist on the new alone but also on the readaptation, perfection and extension of time-worn ideas. This has been true of many of the concepts so prophetically assembled in the slender treatise of Naudeau.

To recapitulate, in the psychiatric practice of today as contrasted with the suggestions of Naudeau, the custom of indiscriminately narcotizing and purging mental patients has been abandoned, not alone by a few leaders, but almost universally. Under the discipline of occupational therapy we have included all the recreations, occupations and arts, but they are now particularized to the needs of the individual patient. We have the right to feel that in today's shock treatment the measured dosage of pharmaceutical administration makes available a graded method inconceivable even ten years ago. Through this control we have attained the possibility of regulating the degree of shock, and of studying the results attainable in variations of the agent and the subject.

Concerning the influence of mental attitudes on physical symptoms, we have good reason to hope that in the near future psychosomatic medicine will learn something more specific than unconvincing variations of "repressed aggression" to account for symptoms ranging from asthma to eczema, and will eventually solve the challenging relationship between glandular secretion and emotional reactions. Finally, through the researches of the psychoanalytic school we have elaborated a theory and a method which places within the reach of less intuitively endowed physicians the possibility of acquiring instruction in the "sublime and difficult art of using resources of psychological approach."

So, in view of the vitality and vigilance existing in psychiatry today this excursion into the past, with its accent on certain principles which

have survived because of their intrinsic merit, may not be trite and futile but may be a source of courage, faith, perseverance and direction for the greatly increasing number of doctors who are to carry on the slow, discouraging struggle against mental misery.

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A REVIEW OF THE MATERNITY STATISTICS OF NEW YORK CITY FOR THE YEAR 1939*

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I. INTRODUCTION

1.) *Definitions.* This discussion will be greatly clarified if certain matters such as definitions, methods of classifying puerperal deaths, and trends are discussed at the outset. The term "puerperal" is used as defined by the Bureau of the Census and includes pregnancy, parturition and lactation. Two terms are currently being used in connection with maternal deaths: "puerperal mortality" and "maternal mortality;" they are frequently used interchangeably. At the New York City Department of Health, the distinction lies in the fact that the *puerperal* mortality rate is more inclusive, since it represents the number of women dying from all conditions directly due to pregnancy and childbirth, including those associated with abortions and ectopic gestations, whereas the *maternal* mortality rate applies to mortality associated with late pregnancy, or in more exact terms, it is the number of women dying from causes associated with pregnancy of 20 weeks or more gestation.

The principle involved in the calculation of a puerperal or maternal mortality rate is that the number of deaths from these causes should be referred to the number of persons exposed to the risk of dying from these causes. In calculating the *puerperal* mortality rate, we take as the numerator of our calculation the number of deaths ascribed to the causes listed in titles 140 to 150 of the International List of Causes of Death, and as our denominator, the number of *single* live births, plus the number of single fetal deaths (reported abortions and still births), the number of multiple births and the number of women who died undelivered. In this paper, we have referred to this denominator as the "reported terminated pregnancies." In calculating the *maternal* mor-

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tality rate, we omit from the numerator, as described above, the number of deaths of women following abortion, ectopic and molar pregnancies and from the denominator the number of pregnancies reported terminated at less than 20 weeks gestation. In the first part of this paper, all of our discussion will refer to "puerperal mortality;" later, "maternal mortality," will be briefly considered.

Unfortunately, in discussing puerperal mortality since the beginning of this century, we are unable to present our material in quite the same manner as we can for 1939. In the past, in keeping with the usual practice, the New York City Health Department based its calculations of puerperal mortality rates upon the number of live births. More recently, the total number of deliveries—live and dead births—reported has been taken as the basis of our rate calculations. This practice differs from the principle enunciated above, in that it omits from the denominator the number of women who die from puerperal causes before delivery and includes the number of infants born alive or dead of multiple pregnancies, instead of the number of such pregnancies. However, for 1939, when all these data were available, such adjustments have been made. We recognize that the numbers described in such terms as "reported terminated pregnancies" or "exposed to risk of dying from puerperal causes" are only approximations to the number of women whose pregnancies have actually been terminated during the course of a given year; live births are not all registered and fetal deaths, especially early abortions, are very incompletely reported. However, these approximations improve from year to year as the reporting of births and fetal deaths becomes more complete. In order to have a comparable figure for our purposes, we shall refer to a "pregnancy rate," which we define as the total number of reported terminated pregnancies per 1,000 population; this rate is subject to the same remarks regarding its incompleteness as is the denominator of the puerperal mortality rate.

2.) *Methods of Classifying Puerperal Deaths.* During the decade 1930 to 1939, deaths associated with pregnancy, childbirth and the puerperal state, were classified under eleven titles (140 to 150, inclusive) of the International List of Causes of Death. In actual practice, in the New York City Department of Health, we have subdivided certain of these titles with the result that our list contains 20 groups, rather than 11, in order that we might obtain information on such items as cesarean section, which would otherwise be lost in a group total.

When only a single cause of death is reported, classification is a relatively simple matter. However, when multiple causes are reported in connection with a death, the choice of the single cause to which that death is to be charged is made in accordance with the rules prescribed by the Bureau of the Census in the manual for the classification of jointly-reported causes of death. The purpose of this joint-cause manual is to make certain that the same combination of causes is classified in the same way by the federal, state and municipal bureaus of vital statistics.

For example, such diseases as syphilis and cancer, and external causes such as accidents, homicide, or suicide, when reported jointly with a puerperal cause, invariably take precedence over the puerperal cause in classification. Pulmonary tuberculosis takes precedence in the assignment over all puerperal causes except those involving septicemia. Death is attributed to chronic nephritis, when this cause is reported jointly with puerperal albuminuria, eclampsia and other toxemias of pregnancy, and certain other and unspecified conditions of the puerperal state. In general, diseases of the puerperal state take precedence over diseases of the heart and circulatory system, pneumonia, influenza, diabetes, and acute nephritis.

All basis for comparing the puerperal death rates in various communities would be destroyed if classification practices were not uniform. If one office were to charge jointly-reported pulmonary tuberculosis and a puerperal condition to tuberculosis while the other charged the same combination of conditions to diseases of the puerperal state, it is apparent that confusion would result.

3.) *Trend of Puerperal Mortality 1901 to 1939.* Before we plunge into the analysis of puerperal mortality for 1939, it is of interest to consider the trend since the year 1901. This information is presented in detail in Table 1 and is summarized in Chart I. Chart I shows that the puerperal mortality rate in New York City remained fairly constant during the years 1901 to 1930 despite war years, "good times," and financial depressions; during that period, the highest puerperal mortality rate was that for 1905 when it was 57.7 per 10,000 total births; the lowest occurred in 1926 when the rate was 45.2. On the whole, all of the rates during the 30 year period clustered about the average of the rates which was just above 50 per 10,000, or 5 per thousand, total births. The graph shows a level trend, with practically no

TABLE 1

CITY OF NEW YORK — PUERPERAL MORTALITY
RATES PER 10,000 REPORTED TERMINATED PREGNANCIES — 1901-1939

Year	Reported Terminated Pregnancies (Live Births and Fetal Deaths)	"Pregnancy Rate" per 10,000 Population	Puerperal Deaths	Puerperal Mortality Rate*
1901	127,750	35.9	648	50.7
1902	133,011	36.3	642	48.3
1903	135,078	35.7	637	47.2
1904	139,288	35.7	727	52.2
1905	141,352	35.1	815	57.7
1906	147,646	35.4	763	51.7
1907	156,351	36.2	783	50.1
1908	153,191	34.3	699	45.6
1909	148,697	32.1	719	48.4
1910	149,751	31.3	761	50.8
1911	147,673	30.3	738	50.0
1912	146,619	29.6	676	46.1
1913	146,629	29.0	668	45.6
1914	147,264	28.7	679	46.1
1915	147,669	28.3	710	48.1
1916	143,917	27.1	653	45.4
1917	147,681	27.3	651	44.1
1918	144,839	26.4	664	45.8
1919	136,361	24.5	644	47.2
1920	139,090	24.5	708	50.9
1921	140,538	24.2	746	53.1
1922	135,809	22.9	699	51.5
1923	135,183	22.3	623	46.1
1924	136,884	22.1	678	49.5
1925	134,924	21.3	697	51.7
1926	131,569	20.4	595	45.2
1927	134,784	20.5	694	51.5
1928	132,453	19.7	668	50.4
1929	130,130	19.0	629	48.3
1930	128,511	18.5	667	51.9
1931	121,200	17.2	698	57.6
1932	115,274	16.2	663	57.5
1933	108,847	15.2	665	61.1
1934	106,309	14.7	492	46.3
1935	105,559	14.5	523	49.5
1936	103,499	14.1	459	44.3
1937	106,835	14.4	408	38.2
1938	107,040	14.3	357	33.4
1939	108,018**	14.3	320	29.6

*Puerperal Mortality Rate is expressed in units of 10,000 "Reported Terminated Pregnancies."

**1939 is the only year in this table for which the item "Reported Terminated Pregnancies" includes the group of women who died of puerperal causes but had not been delivered at the time of death. There were eighty-five such cases reported in 1939. It is also the only year for which adjustments have been made for multiple births.

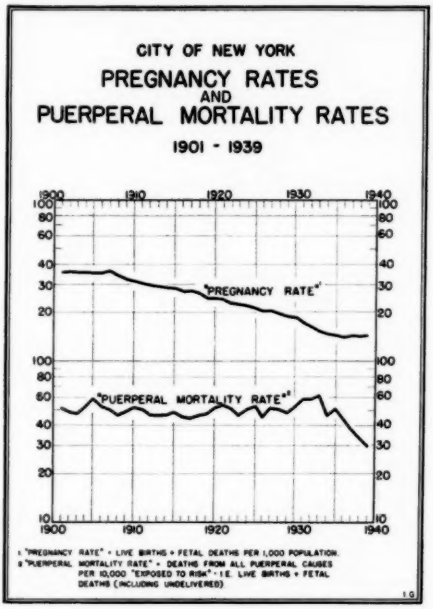
CITY OF NEW YORK
PREGNANCY RATES
AND
PUERPERAL MORTALITY RATES
1901 - 1939
1900
1910
1920
1930
1940
"PREGNANCY RATE"
"PUERPERAL MORTALITY RATE"
1. "PREGNANCY RATE" = LIVE BIRTHS + FETAL DEATHS PER 1,000 POPULATION.
2. "PUERPERAL MORTALITY RATE" = DEATHS FROM ALL PUERPERAL CAUSES PER 10,000 "EXPOSED TO RISK" - I.E. LIVE BIRTHS + FETAL DEATHS (INCLUDING UNDELIVERED)

Chart I

slope, and only minor fluctuations during the entire thirty years. From 1930 to 1933, when the economic depression was apparently having its greatest effect upon living standards, there was an increase in puerperal mortality in New York City. In these four years, the death rates were, in round numbers, 52, 58, 58 and 61.

It is of interest to note that the "pregnancy rate," i.e., the number of reported terminated pregnancies per thousand of population, which is plotted on the same chart, drops progressively from 1901 to 1933, but that the decline is accompanied by relatively little change in the puerperal mortality rate.

The picture changes rather abruptly in 1933. From 1933 to 1939, there is a sharp drop in puerperal mortality from 61.1 to 29.6 per 10,000 reported terminated pregnancies. The pregnancy rate, on the other hand, remains fairly level during that seven-year period. Little explanation can be offered for the behavior of the pregnancy rate, but several hypotheses may be advanced concerning the recent, rather spectacular, decline in puerperal mortality.

4.) *Factors in the Decline of Puerperal Mortality Since 1933.* In New York City, home relief authorities include a great deal of welfare work in their activities. They supervise their pregnant clients to the extent of providing for special diet, paying for ante- and post-partum care if arrangements are made to have the delivery at home, and make many practical suggestions for indigent pregnant women. It is highly probable that many underprivileged persons now fare considerably better under intelligently-administered welfare work than they did on their own responsibility in the past. Moreover, the fact that the "pregnancy rate" has remained practically unchanged during the years since 1933 suggests that a stabilization of the birth rate at a favorable level may have occurred at about 1934. It is rather difficult for us to discuss pregnancy in connection with economic factors because of the paucity of data available, but discussion later in this paper suggests economic status as a possible influence upon the puerperal mortality rate.

A more tangible element in the reduction of puerperal mortality is the action taken by organized medical groups within the last decade. In 1933, The New York Academy of Medicine issued its report on "Maternal Mortality in New York City"; this comprehensive survey did more than anything else to focus medical attention upon the problem. Commencing in 1936, the medical societies of the Counties of Kings, Queens and New York arranged with the Department of Health to obtain information regarding women who died from puerperal causes in order to conduct local studies of the problem. Questionnaires were sent to the physicians reporting puerperal deaths, and these inquiries formed the basis of discussion in committee meetings about the "preventability" of these deaths, while preserving, in open meetings, the anonymity of patient, doctor and hospital. The Bronx County Medical Society had for many years been independently active in attempting to reduce maternal mortality. This concerted planning toward a common goal has certainly resulted in greater emphasis upon prenatal care and improvement in obstetric practices and facilities. It is practically impossible to measure the effects of these efforts of the medical profession upon the declining puerperal mortality, but the fact that puerperal deaths are decreasing concurrently with this specific action strongly suggests some causal connection. In fact, all investigation into the causes of reduction of puerperal mortality at this time must involve an element of speculation. With improvement in record

keeping and statistical analysis, we may anticipate a more scientific approach to the problem in the near future.

II. Source of Raw Material for this Study

Early in 1938, the Bureau of Records of the New York City Department of Health initiated a procedure to make vital records more valuable than heretofore in current public health practice. This procedure, which has been endorsed by the Children's Bureau, and was widely adopted by state health departments when new forms were adopted for use commencing in 1940, consists in requesting in a confidential medical report on the back of birth and fetal death certificates, information concerning measurements of the baby, complications of pregnancy, and important facts about the delivery. Several minor changes were made in the content of these confidential reports as a consequence of the early period of trial, and the information requested in the supplementary medical report of the newest form of fetal death certificate (which includes all questions asked in connection with live births) appears in Figure 1. On January 1, 1939, reporting of this confidential information became compulsory. The death certificate, which was previously our only source of information, usually yielded relatively little information of interest in a study of puerperal mortality. Consequently, in 1939, for the first time, we were able to extract from the combined use of birth, fetal death and death certificates, data that would serve as an excellent basis for the establishment of routine analyses of puerperal deaths.

III. Size and Character of the Sample

In New York City in 1939, deaths with reference to pregnancy and childbirth totaled 365—a figure fairly easy to remember. This number includes 14 criminal abortions, 31 deaths in which the puerperal condition was reported as a *contributory* cause, and 320 deaths, which, under the rules for the classification of jointly-reported causes and the International List, were charged to diseases and conditions of pregnancy, childbirth, and the puerperal state.

Seven of the deaths charged to criminal abortion occurred in Manhattan, two each were reported from Bronx, Brooklyn, and Queens, and one from Richmond. Nine of these women were white, and of these nine, four were reported as single; the marital condition of one

CITY OF NEW YORK

BACK OF

STILLBIRTH (FETAL DEATH) CERTIFICATE

SUPPLEMENTARY MEDICAL REPORT—Confidential
(Only for scientific purposes approved by the Board of Health)

18c. Number born dead PREVIOUS to this pregnancy, including abortions.....

20. Period of gestation.....weeks.

21. *Length at birth (crown to heel).....centimeters or.....inches.

22. *Weight at birth.....grams or.....lbs.....ounces.

23. Was pregnancy normal?.....

24. If not, specify complications†.....

25. Did mother have serological test for syphilis during THIS pregnancy?.....If so, date.....

26. Result.....If no test, give reason.....

27. *Total duration of labor.....hours.

28. Was labor (abortion) induced?.....

29. If so, specify method of induction†.....

30. Were any operative or instrumental procedures used?.....
Specify†.....

31. Indications for procedures†.....
.....

32. *Did fetal death occur: before or during labor?.....
If during labor, give stage.....
before or during operation?.....

33. Was fetus macerated?.....

34. Was autopsy performed?.....If so, give date.....
By whom?.....

35. Cause of fetal death?†
(a) Determined in the fetus.....
(b) In mother.....

* Not required to be answered in ectopic pregnancy or abortion of less than 20 weeks gestation.
† See inside of front cover.

Gestation		
Length		
Weight		
Pregnancy		
Sero. Test		
Duration	Labor	
Induced	Labor	
Type	Operation	
Indication	Operation	
Labor	Time of Death	
Operation	Time of Death	
Macerated		
Autopsy		
Fetus	Cause of Death	
Mother	Cause of Death	

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TABLE 2

CITY OF NEW YORK
DEATHS WITH PUERPERAL CONDITIONS AS SECONDARY CAUSES—1939

International List Number	Primary Cause of Death	Number of Deaths		
		Total	White	Colored
	Total	31	23	8
8	Scarlet Fever	1	1	0
23	Pulmonary Tuberculosis	4	1	3
34	Syphilis (acquired)	2	0	2
46-50	Cancer	4	4	0
90-95	Heart Disease	7	7	0
108	Lobar Pneumonia	1	1	0
121	Appendicitis	1	1	0
125	Yellow Atrophy of Liver	2	2	0
131	Chronic Nephritis	4	3	1
179	Accidental Poisonings	2	1	1
186-6	Falls at home	3	2	1

was not stated. Of the four colored women, one was reported unmarried. Under revision of the International List, used during 1939, criminal abortions were classified among the homicides. These 14 deaths, therefore, will receive little further consideration in this paper.

The classification of the 31 deaths in which conditions of pregnancy and childbirth were mentioned as a secondary cause is shown in Table 2. These deaths are not treated as puerperal deaths, and they are dismissed from further consideration in this paper.

Then, for the purposes of this analysis, we shall consider certain pertinent facts concerning the 320 deaths reported in 1939 in New York City as resulting primarily from puerperal causes. As soon as one of these deaths was reported, a search was made to determine whether the puerperal death was associated with a live birth, a fetal death or whether the mother died undelivered. If the delivery was not recorded in a live birth or a fetal death certificate, attempts were made to ascer-

TABLE 3

CITY OF NEW YORK — PUERPERAL MORTALITY — BY COLOR AND BOROUGH OF OCCURRENCE
NATURE OF TERMINATION OF PREGNANCY — 1939

Borough of Occurrence	Total Reported Terminated Pregnancies			Nature of Termination of Pregnancy											
				Single Live Births			Single Dead Births			Multiple Births Live or Dead			Undelivered or Unrecorded		
	T	W	C	T	W	C	T	W	C	T	W	C	T	W	C
Entire City	108018	99559	8459	100190	92552	7638	6594	5905	689	1149	1048	101	85	54	31
Manhattan	35300	30453	4847	32591	28256	4335	2319	1888	431	345	287	58	45	22	23
Bronx	15639	15056	583	14339	13796	543	1122	1087	35	172	167	5	6	6	0
Brooklyn	42346	39836	2510	39464	37177	2287	2390	2206	184	470	439	31	22	14	8
Queens	12289	11854	435	11477	11084	393	669	633	36	133	127	6	10	10	0
Richmond	2444	2360	84	2319	2239	80	94	91	3	29	28	1	2	2	0

tain whether delivery had been effected prior to death, and physicians were requested to submit reports in appropriate cases. By this matching of puerperal deaths with birth and fetal death certificates, it was found that of the 320 women who died from puerperal causes, 146 were associated with live births, 89 with fetal deaths, and 85 comprise a group, many of whom were known to have died undelivered; for the remainder—mostly deaths charged to early abortion—no information regarding delivery was available. Since the supplementary information regarding pregnancy was available only through birth and fetal death certificates, some of our discussion will cover only the 235 cases for which deliveries were recorded. It is admitted that this is, in general, a rather limited sample, but caution will be exercised throughout the discussion in drawing conclusions based on small figures.

On the other hand, our control group, the number of women who survived pregnancy, is huge compared to the mortality group. In 1939, reports were received of 108,018 terminated pregnancies, for which data are available for purposes of analysis and study. These pregnancies resulted in 100,190 single live births, 6,594 single dead births, 1,149 multiple births, and 85 puerperal deaths for which no delivery was recorded by the filing of a birth or fetal death certificate. The nature of termination of pregnancy, by color and borough of occurrence is presented in Table 3.

IV. Geographic Distribution of Puerperal Deaths, by Color

To meet the needs and interests of local professional groups, puerperal mortality has been investigated according to the borough of occurrence of the puerperal death. It might be mentioned parenthetically that the borough of occurrence of death differed in only two cases from the borough of delivery. This does not affect our discussion in any way and will not be referred to again.

In 1939, for the entire city, as stated above, there were 320 deaths attributed primarily to puerperal causes; of these, 64, exactly one-fifth, were colored, and the remaining 256, or four-fifths, were white. On the basis of these figures, *the puerperal mortality rate for the city as a whole was 29.6 per 10,000 reported terminated pregnancies.* Detailed information on these points, by borough and color, is presented in Table 4.

As may be expected on the basis of experience, there is a wide

TABLE 4

CITY OF NEW YORK — PUERPERAL MORTALITY
BY COLOR AND BOROUGH OF OCCURRENCE
RATES PER 10,000 REPORTED TERMINATED PREGNANCIES — 1939

Borough of Occurrence	Reported Terminated Pregnancies			Number of Puerperal Deaths			Puerperal Death Rate Per 10,000 Reported Terminated Pregnancies		
	T	W	C	T	W	C	T	W	C
Entire City	108,018	99,559	8,459	320	256	64	29.6	25.7	75.7
Manhattan	35,300	30,453	4,847	115	74	41	32.6	24.3	84.6
Bronx	15,639	15,056	583	43	42	1	27.5	27.9	*
Brooklyn	42,346	39,836	2,510	110	91	19	26.0	22.8	75.7
Queens	12,289	11,854	435	44	41	3	35.8	34.3	*
Richmond	2,444	2,360	84	8	8	0	32.7	33.9	—

*Numbers too small to yield statistically significant rates.

difference between the rate for the white group, which was 25.7, and the colored group, which was 75.7. Before puerperal mortality is considered on a borough basis, it should be pointed out that the proportions of white and colored in the population, and the reported terminated pregnancies, by color, vary markedly among the five boroughs. To correct for this, a puerperal mortality rate was calculated for each borough taking this variation into account. Of the total number of reported terminated pregnancies for the city as a whole, 92.2 per cent were white, and 7.8 per cent were colored. The percentages of the colored in the total terminated pregnancies in the several boroughs in 1939, were as follows: Manhattan 13.7, Bronx 3.7, Brooklyn 5.9, Queens 3.5 and Richmond 3.4. In order to secure a properly weighted rate for each borough, allowing for differences in color distribution, new rates were computed on the hypothesis that each borough had the same proportion of colored pregnancies as the city as a whole. For example, the puerperal mortality rates for white and colored in Brooklyn were 22.8 and 75.7, respectively. Multiplying the white rate by .922 and the colored by .078, gives 21.02 and 5.90. Adding these two rates together gives Brooklyn a "corrected" puerperal death rate of 26.9. The crude color-specific, and "corrected" rates

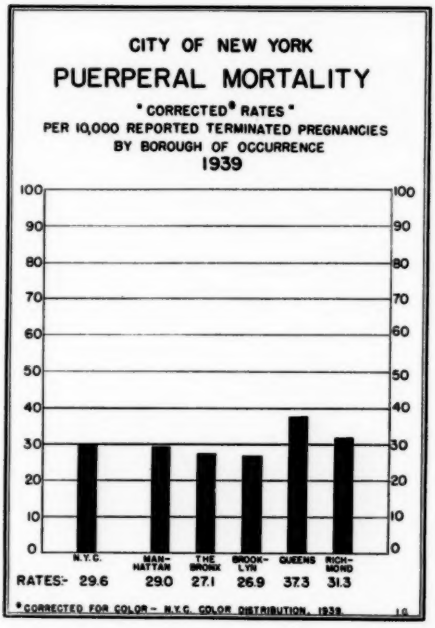

Chart II

TABLE 5

CITY OF NEW YORK—PUERPERAL MORTALITY BY COLOR AND BOROUGH
CRUDE, COLOR-SPECIFIC AND CORRECTED RATES — 1939

Borough	Crude Puer. Death Rate	Color Specifications		Col. 2 × 92.2*	Col. 3 × 7.8*	"Corrected" Rate Col. 3 + Col. 4
		White	Colored			
	1.	2.	3.	4.	5.	6.
Entire City	29.6	25.7	75.7	23.70	5.90	29.6
Manhattan	32.6	24.3	84.6	22.40	6.60	29.0
Bronx	27.5	27.9	**	25.72	1.34	27.1
Brooklyn	26.0	22.8	75.7	21.02	5.90	26.9
Queens	35.8	34.6	**	31.90	5.38	37.3
Richmond	32.7	33.9	0.0	31.26	0.0	31.3

*Color Distribution—Reported Terminated Pregnancies—New York City, 1939.
White — 92.2% Colored — 7.8%
**Numbers too small to yield statistically significant rates.

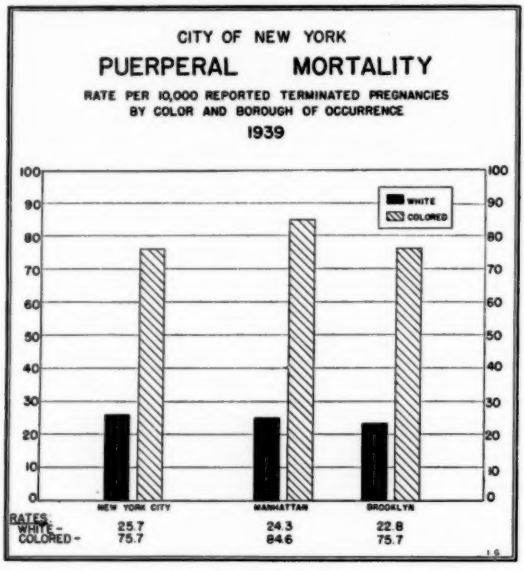

Chart III

for each borough are shown in Table 5 and the corrected rates are shown in Chart II.

Although this correction for color, changes the rates to an extent, it may be observed that the ranking of the boroughs according to the order of *puerperal* mortality rate is not changed. Both before and after the weighting process, Brooklyn has the lowest rate, Bronx the next lowest, and then Manhattan, Richmond and Queens follow in ascending order. Most of the puerperal deaths among the colored occurred in Manhattan and Brooklyn, where the specific rates for the colored were 84.6 and 75.7, respectively, as compared with 24.3 and 22.8, respectively, for the white groups. Richmond had no puerperal deaths among the colored, Queens only 3, and Bronx only one. The terminated pregnancies among the colored in these two boroughs were so few that rates have not been calculated because they would be statistically insignificant. Chart III shows the puerperal mortality rates for the white and colored in the city as a whole and for Manhattan and Brooklyn.

TABLE 6

CITY OF NEW YORK—DEATHS FROM PUERPERAL CAUSES BY NATURE OF TERMINATION OF PREGNANCY—BOROUGH AND COLOR—1939

Borough of Occurrence	Total Puerperal Deaths			Puerperal Deaths Associated With								
				Live Births			Fetal Deaths			Undelivered or Unrecorded Delivery		
	T	W	C	T	W	C	T	W	C	T	W	C
Entire City	320	256	64	146	126	20	89	76	13	85	54	31
Manhattan	115	74	41	42	30	12	28	22	6	45	22	23
Bronx	43	42	1	22	22	0	15	14	1	6	6	0
Brooklyn	110	91	19	52	46	6	36	31	5	22	14	8
Queens	44	41	3	27	25	2	7	6	1	10	10	0
Richmond	8	8	0	3	3	0	3	3	0	2	2	0

V. NATURE OF TERMINATION OF PREGNANCY ASSOCIATED WITH PUERPERAL DEATH

It has been previously mentioned that of the 320 puerperal deaths, 146 were associated with live births, 89 with fetal deaths, and that there were 85 for which no birth or fetal death was registered. The last group of 85 women includes some who actually died undelivered as well as others for whom the result of the delivery was not recorded. Data for each borough, by color, are presented in Table 6.

Referring the 146 maternal deaths associated with live births, to the pregnancies that terminated with a live-born child (101,176), we have a puerperal death rate of 14.4 per 10,000. If we make a similar calculation of the puerperal deaths associated with fetal deaths–abortions and stillbirths–we obtain a rate of 131.7 per 10,000. It, therefore, *appears* that the hazard to the mother's life is much greater when pregnancy does not end in a live birth. Exactly how much greater this hazard is, it is difficult to say because of the incompleteness in reporting early abortions. It also appears that the mortality of women from puerperal causes before delivery is important, although we lack complete information on this point. Attention is called to the large number of such cases in Manhattan.

TABLE 7

CITY OF NEW YORK — AGE DISTRIBUTION OF MOTHERS BY COLOR — 1939

Age Groups	Mothers Surviving						Mothers Dying From Puerperal Causes					
	Number*			Per Cent			Number			Per Cent		
	T	W	C	T	W	C	T	W	C	T	W	C
All Ages	107665	99270	8395				320	256	64			
Age not stated	189	147	42									
Total, age stated	107476	99123	8353	100.0	100.0	100.0	320	256	64	100.0	100.0	100.0
Less than 15 yrs.	52	12	40	0.0	0.0	0.5						
15-19	5223	3746	1477	4.9	3.8	17.7	9	3	6	2.8	1.2	9.4
20-24	27027	24037	2990	25.1	24.2	35.8	58	41	17	18.1	16.0	26.6
25-29	37359	35499	1860	34.8	35.8	22.3	76	60	16	23.8	23.4	25.0
30-34	24997	23777	1220	23.3	24.0	14.6	92	80	12	28.8	31.3	18.7
35-39	10285	9673	612	9.6	9.8	7.3	63	54	9	19.7	21.1	14.1
40-44	2362	2226	136	2.2	2.2	1.6	19	16	3	5.9	6.2	4.7
45-49	167	149	18	0.1	0.2	0.2	2	2	0	0.6	0.8	0.0
50-54	4	4	0	0.0	0.0	0.0	1	0	1	0.3	0.0	1.6

*In addition, there were 33 fetal deaths with the color and age of mother unknown.

VI. Age Distribution of Mothers

1.) *Percentage Distribution of Fatalities and Survivors.* It will be of interest to compare the age distribution of those women who died from puerperal causes with that of women who survived pregnancy in the city during the year. Of course, the latter group, comprising 107,665 women, is much larger than the mortality group of only 320 individuals. Allowance should be made for this great difference in comparing the two groups; conclusions based on the larger group will be statistically more reliable. These facts are presented in Table 7 and Chart IV.

In the graph on the left side of Chart IV, the heavy line shows the *per cent* of the 99,559 *white* women whose pregnancies were reported terminated in 1939, falling in each age group; the dashed line gives the same information for white women who died from a puerperal cause. The contours of the two polygons are generally similar in shape, but the points of the curve for the "mortality" group appear to be consistently one five-year unit higher than those of the "surviving" group. The 25 to 29 year age period is the peak age for "surviving" women, with 35.8 per cent falling in that age group. The peak age period for white women who died of puerperal causes is from 30 to 34 years; this age group accounts for 31.3 per cent of these women. This graph, for white women, shows that there is a decided tendency for those who died from puerperal causes to fall in older age groups than those who survived. In other words, the hazards of pregnancy appear greater with increasing age.

The details of the picture are somewhat different for colored women. In discussing them, it should be emphasized that only 64 women are involved in the "mortality" sample, and consequently we may expect erratic behavior in the percentages computed on this small base. The most distinguishing feature of the two curves on the right of Chart IV is that the peak age group for the women who survived and those who died is the same: 20 to 24 years. Also, the peak of both colored groups is lower than that for the corresponding white groups. As is the case for the white women, the percentages of colored women who died are smaller than those for the survivors below the peak age, and larger above it. Even this casual inspection of the graph leads to the conclusion that the women who died from puerperal causes were older than those who survived.

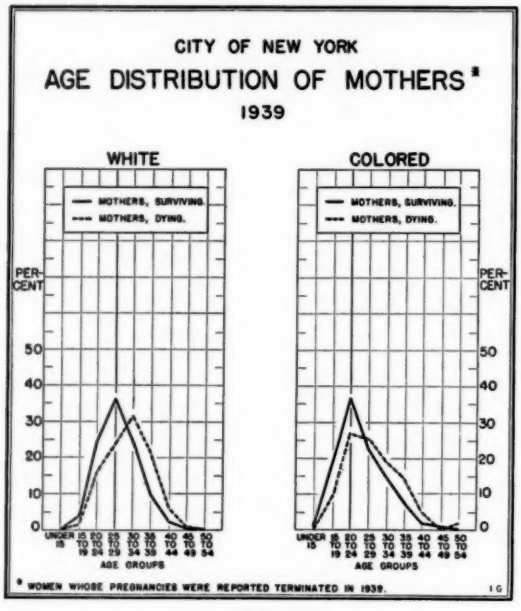

Chart IV

2.) *The Median Ages of Fatalities and Survivors.* This fact will be even more evident if we inspect the median age for each of these groups by color.

	Total	White	Colored
Median age of "surviving" women.....	27.9	28.1	24.5
Median age of women who died.....	29.9	30.5	26.9

The median age for women whose pregnancies terminated in 1939, and who survived, is 27.9; the median age for the mortality group, 29.9, is exactly two years higher. For white women who survived, the median age was 28.1 as compared with 30.5 for white women who died. Although a difference of 2 years is numerically small, it should be borne in mind that we are considering a limited age "range"—from about 15 to about 45 years—and that such a difference is probably of some importance.

For colored surviving women, the median age was 24.5 years; for those who died, the median was 26.9 years. Although all colored

women whose pregnancies terminated in 1939 are, in general, younger by about 4 years (according to the median ages) than white women, the general tendency for colored women who died toward older age groups is apparent from inspection of the graph. As is the case for the white women, percentages of colored women who died are smaller than those for survivors below the peak age, and larger above it.

To summarize briefly: (1) Colored women whose pregnancies were reported terminated are younger by about 4 years than white women. (2) Women whose pregnancies were reported terminated and who survived are younger by about 2 years than those who died. This observation holds for both white and colored.

3.) *Puerperal Fatality Rates by Age.* The increasing hazard of pregnancy as age advances is more clearly shown in the case fatality rates by age. In making this calculation, we take the number of women dying of a puerperal cause in each age group and divide that number by the number of pregnancies terminated at that age. The details of this are presented in Table 8 and the results are shown graphically in Chart V.

Contrary to the usual impression, there is no evidence in these data of high mortality associated with pregnancy at early ages. It is true that there were only 52 pregnancies reported terminated below 15 years of age—12 among white girls and 40 among colored—but no puerperal deaths were reported in this age group. Deathrates are lowest in the age period 15 to 19 years, and appear to increase irreversibly with age, reaching their maxima in the age group 40 to 44 years.

VII. Puerperal Mortality by Place of Occurrence

It is with some hesitation that I present the data with regard to puerperal mortality by place of occurrence because they are so susceptible of being misinterpreted.

The type of institution may perhaps be taken as an indication of the social and economic status of its clientele. In general, the municipal hospitals may be said to receive persons from the lowest economic levels —the undernourished, the sickly, the relief client. Furthermore, emergency cases, with their complications, are taken to municipal hospitals by ambulances, as a general rule. The voluntary hospitals receive—on the average—persons from a higher economic level; some can pay their entire charges, others a part, while still other patients in some of these

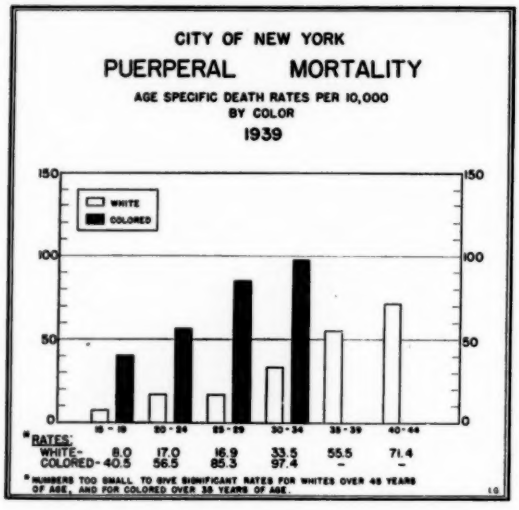

Chart V

TABLE 8

CITY OF NEW YORK — DEATHS FROM PUERPERAL CAUSES
MORTALITY BY COLOR AND AGE GROUPS — 1939

Age Groups	Mothers in Age Group			Puerperal Deaths in Age Group			Puerperal Death Rate per 10,000		
	T	W	C	T	W	C	T	W	C
All Ages	108018*	99526	8459	320	256	64	29.6	25.7	75.7
Age not stated	222*	147	42						
Total, age stated	107796	99379	8417	320	256	64	29.8	25.8	76.0
Less than 15 years	52	12	40	0	0	0	—	—	—
15 - 19	5232	3947	1483	9	3	6	17.2	8.0	40.5
20 - 24	27085	24078	3007	58	41	17	21.4	17.0	56.5
25 - 29	37435	35559	1876	76	60	16	20.3	16.9	85.3
30 - 34	25089	23857	1232	92	80	12	36.7	33.5	97.4
35 - 39	10348	9727	621	63	54	9	60.9	55.5	**
40 - 44	2381	2242	139	19	16	3	79.8	71.4	**
45 - 49	169	151	18	2	2	0	**	**	—
50 - 54	5	4	1	1	0	1	**	—	**

*Includes 33 fetal deaths with the color and age of mother unknown.
**Numbers too small to yield statistically significant rates.

TABLE 9

CITY OF NEW YORK—DEATHS FROM PUERPERAL CAUSES BY PLACE OF DEATH AND TYPE OF INSTITUTION—1939

Place of Death—Type of Institution	Number*	Total Reported Terminated Pregnancies			Number of Puerperal Deaths			Puerperal Death Rate		
		T	W	C	T	W	C	T	W	C
New York City—Total		108018	99559	8459	320	256	64	29.6	25.7	75.7
Municipal Hospitals	17	18639	13470	5169	119	67	52	63.8	49.7	100.6
Voluntary Hospitals—City Aid	57	50278	48169	2109	127	119	8	25.3	24.7	37.9
Voluntary Hospitals—No City Aid	21	9463	9443	20	24	24	0	25.4	25.4	—
Proprietary Hospitals	63	21318	21193	125	38	37	1	17.8	17.5	**
Other Government Institutions	7	231	228	3	0	0	0	—	—	—
Home		7952	6953	999	12	9	3	15.1	12.9	30.0
Unknown		137	103	34	0	0	0	—	—	—

*Number of hospitals from which at least one terminated pregnancy was reported.
**Number too small to yield a statistically significant rate.

institutions have their expenses paid, in whole or in part, by the city, by church funds or by other philanthropic agencies. Unfortunately, we are unable to determine from the material available to us whether the persons who died in voluntary hospitals were recipients of financial assistance; knowledge of the source of hospital funds is not, in itself, sufficient indication of the economic status of individual patients in voluntary hospitals. The proprietary hospitals cater exclusively to those who can pay their own way in full. The government institutions are station hospitals in Army, Navy and Marine Corps reservations. Insofar as maternity care is concerned, practice in these institutions is restricted to wives of commissioned and non-commissioned officers—people who have permanent employment and good food, and are well housed.

Those cases for which the place of termination of pregnancy was unknown are mostly comprised of stillborn fetuses found in ash cans, vacant lots, and similar places.

With these facts in mind, these data are presented in Table 9.

TABLE 10

CITY OF NEW YORK — PUERPERAL MORTALITY
IN MUNICIPAL HOSPITALS BY COLOR AND BOROUGH
RATE PER 10,000 REPORTED TERMINATED PREGNANCIES — 1939

Borough	Total	White	Colored
New York City — Total	29.6	25.7	75.7
Municipal Hospitals — Total	63.8	49.7	100.6
Manhattan	93.6	67.4	118.6
Bronx	58.6	64.6	*
Brooklyn	47.3	36.7	91.2
Queens	38.2	30.7	*
Richmond	—	—	—

*Numbers too small to yield statistically significant rates.

VIII. CAUSES OF PUERPERAL DEATH

1.) *The Trend of Puerperal Mortality–By Cause–1931 to 1939.* In the decade ending with 1939, the puerperal death rate declined approximately 50 per cent; the rate for 1930 was 57.8 per ten thousand reported terminated pregnancies, for 1939 it was 29.6. Certain causes such as hemorrhage, septicemia and accidents of childbirth (which include deaths following cesarean section, dystocia, trauma of the uterus and adjacent organs, and other accidents of childbirth) have lagged behind the general downward trend while deaths charged to abortion, toxemia and a group of miscellaneous causes have shown a greater reduction than puerperal diseases as a whole.

As a matter of fact, the 1939 death rate from puerperal septicemia–not including sepsis following abortion or ectopic gestation–was only about 16 per cent lower than the 1930 rate. The increase in the 1939 rate after the low established in 1938 is surprising in view of widespread use of sulfanilamide which was hailed as a cure for so many infections.

From this table, we see that the mortality from puerperal causes among the white clients of the municipal hospitals–49.7 per 10,000 reported terminated pregnancies–is twice as high as for the average white woman in voluntary hospitals, almost three times as high as the

rate among her sisters in proprietary hospitals and almost four times as high as among the women who had their babies at home. In making this latter observation, it must be borne in mind that many obstetrical cases that run into difficulty at home, are rushed to a hospital in a critical condition, and if the woman dies that death is charged against the hospital. In general, it is the easy, uncomplicated case that remains at home.

Unfortunately, the experience among negro women is quite limited. But here also, the puerperal death rate in the municipal hospitals—100.6 per 10,000 reported terminated pregnancies—was almost three times as high as experienced in the group of voluntary hospitals that received city aid, and *more* than three times as high as that among women who remained at home.

One of the factors in the favorable showing made by the Borough of Brooklyn in puerperal mortality in 1939 was the low rate among white women treated in municipal hospitals. This is evident from the figures in Table 10.

Hemorrhage was the cause to show the next strongest resistance to reduction; the death rate from this cause was only one-third lower in 1939 than it was in 1930.

The mortality classified as "accidents of pregnancy," a term which includes the mortality ascribed to cesarean section, showed a drop of approximately 45 per cent—just slightly less than the average for the whole puerperal group.

On the other hand, the deaths charged to abortion have declined slightly more than 50 per cent, deaths from toxemia more than 60 per cent and deaths from the miscellaneous group of puerperal causes, not elsewhere classified, dropped about two-thirds between 1930 and 1939. These facts are presented in Table 11 and are shown graphically in Chart VI.

2.) *The Causes of Puerperal Mortality in New York City during 1939.* The numbers of puerperal deaths in New York City during 1939, by cause are shown in Table 12.

During that year, more puerperal deaths were attributed to septicemia—title 145 of the International List—than to any other single puerperal cause. The 74 deaths charged to this cause comprise slightly more than 23 per cent (23.1) of all the deaths charged to puerperal causes. These figures, however, do not include deaths from septic abortion or ectopic gestation with sepsis. The mortality rate from deaths ascribed

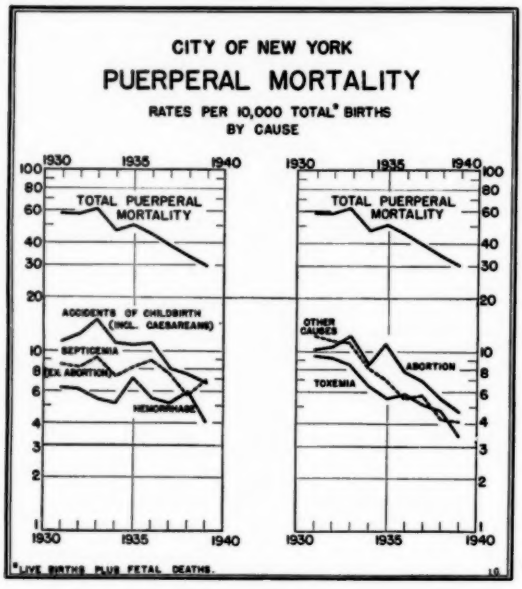

Chart VI

TABLE 11

CITY OF NEW YORK — PUERPERAL MORTALITY BY CAUSE
RATES PER 10,000 TOTAL BIRTHS — 1931 - 1939

Year	Total (140-150)	Abortion (largely with sepsis) (140-141)	Hemorrhage (144)	Septicemia (not Abortion) (145)	Toxemia (146-147)	Accidents of Childbirth (including Cesareans) (149)	All Others (142, 143, 148, 150)
1931	57.8	10.2	6.2	8.4	9.4	11.3	12.2
1932	57.5	10.5	6.1	8.1	9.2	12.3	11.5
1933	61.1	12.2	5.4	9.2	8.4	14.8	11.2
1934	46.3	8.5	5.1	7.2	6.4	11.1	8.1
1935	49.5	11.1	7.0	8.1	5.5	10.8	6.9
1936	44.3	7.8	5.5	8.8	5.8	11.0	5.5
1937	38.2	6.8	5.1	7.5	5.1	8.0	5.7
1938	33.4	5.5	5.9	5.7	4.7	7.4	4.2
1939*	29.6	4.6	4.0	6.8	3.4	6.7	4.1

*Rates for the year 1939 are based upon total reported terminated pregnancies, which include single live births, single fetal deaths, multiple births, and those women who died undelivered.

TABLE 12

CITY OF NEW YORK — PUERPERAL MORTALITY BY CAUSE RATES PER 10,000 REPORTED TERMINATED PREGNANCIES — 1939

Cause— International List Numbers and Titles	Deaths from Puerperal Causes						Puerperal Mortality Rate		
	Number			Per Cent					
	T	W	C	T	W	C	T	W	C
140-150. Total diseases of pregnancy, childbirth and the puerperal state.	320	255	65	100.0	100.0	100.0	29.6	25.6	76.8
140. Abortion with septic conditions	48	29	19	15.0	11.4	29.2	4.4	2.9	22.5
141. Abortion without mention of septic conditions	2	2	0	0.6	0.8	—	0.2	0.2	—
142[1] Ectopic gestation with septic conditions specified	5	4	1	1.6	1.6	1.5	0.5	0.4	1.2
142[2] Ectopic gestation without mention of septic conditions	15	11	4	4.7	4.3	6.2	1.4	1.1	4.7
143. Other accidents of pregnancy	4	4	0	1.3	1.6	—	0.4	0.4	—
144[1] Placenta praevia	16	15	1	5.0	5.9	1.5	1.5	1.5	1.2
144[2] Other puerperal hemorrhages	27	23	4	8.4	9.0	6.2	2.5	2.3	4.7
145[1] Puerperal septicemia and pyemia	73	56	17	22.8	21.9	26.1	6.7	5.7	20.1
145[2] Puerperal tetanus	1	1	0	0.3	0.4	—	0.1	0.1	—
146. Puerperal albuminuria and eclampsia	24	21	3	7.5	8.2	4.6	2.2	2.1	3.5
147. Other toxemias of pregnancy	13	9	4	4.1	3.5	6.2	1.2	0.9	4.7
148. Puerperal phlegmasia alba dolens, embolus, sudden death (not specified as septic)	18	15	3	5.6	5.9	4.6	1.7	1.5	3.5
149[1] Cesarean operation	24	22	2	7.5	8.6	3.1	2.2	2.2	2.4
149[2] Trauma of uterus and adjacent organs	11	8	3	3.4	3.1	4.6	1.0	0.8	3.5
149[3] Dystocia and abnormal presentations	19	19	0	5.9	7.5	—	1.8	1.9	—
149[4] Other and ill-defined accidents of childbirth	18	14	4	5.6	5.5	6.2	1.7	1.4	4.7
150. Other and unspecified conditions of puerperal state	2	2	0	0.6	0.8	—	0.2	0.2	—

to septicemia was 6.8 per 10,000 reported terminated pregnancies—5.8 among white women and 20.1 among the colored. If all puerperal deaths in which infection was a factor were grouped together (74 from septicemia, 48 from septic abortions, and 5 from ectopic gestation with sepsis) we see that infection was present in 127, or approximately 40 per cent of the 320 puerperal deaths.

The conditions grouped under the title 149—other accidents of childbirth—were next in importance to septicemia. The total number of deaths charged to accidents of childbirth was 72, or 22.5 per cent of the total. The mortality rate was 6.7 per ten thousand—6.3 among whites and 10.6 among colored. These deaths include 24 charged to cesarean operations, which will be more fully discussed later, 8 charged to trauma of the uterus and adjacent organs, 19 charged to dystocia and abnormal presentations, and 14 charged to other and ill-defined accidents of childbirth.

Abortion—titles 140 and 141—was charged with 50 deaths, or 15.6 per cent of the mortality from puerperal causes during the year. All but 2 of these 50 deaths were due to septic abortions. The death rate from abortion was 4.6 per 10,000 pregnancies reported terminated during the year—3.1 among white women and 22.5 among the colored. The rate among the colored was about seven times as high as among white women. If the 14 deaths ascribed to criminal abortion were combined with this group, we find that it stands next to septicemia in importance. It is suspected that there are more deaths due to criminal abortion than are reported as such; those deaths are probably reported as due to septic abortion.

Hemorrhage—title 144—was given as the cause of 13.4 per cent of puerperal deaths, ranking fourth in the causes of puerperal mortality. The mortality rate was 4.0 per 10,000 for all pregnancies reported terminated—3.8 among white women and 5.9 among the colored. Of the 43 deaths charged to hemorrhage, 16, or more than one-third were due to placenta previa.

Toxemia of pregnancy—titles 146 and 147, combined—at the beginning of the decade, outranked both septicemia and hemorrhage as a cause of puerperal death; in 1939, it was charged with only 11.6 per cent of the total puerperal mortality and the corresponding rate was 3.4 per 10,000 pregnancies reported terminated. Among white women, the rate was 3.0; among colored 8.2. Twenty-four of the deaths ascribed

TABLE 13

CITY OF NEW YORK—DEATHS FROM PUERPERAL CAUSES SHOWING SECONDARY CAUSES OF DEATH—1939

Secondary Cause of Puerperal Death International List Numbers and Titles	Primary Cause of Puerperal Death							
	Total—All Causes 140-150	Abortions—(largely with sepsis) 140-141	Hemorrhages 144	Septicemia (excluding abortions) 145	Toxemias 146-147	Cesareans 149[1]	Accidents of Childbirth 149[2-4]	All Other Causes 142, 143, 148, 150
Total	320	50	43	74	37	24	48	44
Puerperal Secondaries—Total	92	4	21	36	8	7	10	6
Abortions (140-141)	1	—	0	0	1	0	0	0
Hemorrhages (144)	7	2	—	2	0	0	2	1
Septicemia (145)	0	0	0	—	0	0	0	0
Toxemias (146-147)	10	0	3	6	—	0	1	0
Cesareans (149[1])	35	0	10	20	4	—	1	0
Accidents of Childbirth (149[2-4])	17	0	6	5	3	2	—	1
Other Puerperal Causes (142, 143, 148, 150)	22	2	2	3	0	5	6	4
Non-Puerperal Secondaries—Total	80	10	3	20	6	11	19	11
Non-malignant Uterine Tumors (54[2])	5	1	1	1	0	2	0	0
Diseases of Circulatory System (90-103)	14	0	0	3	0	5	4	2
Pneumonia and Influenza (11, 107-109)	18	2	0	3	1	2	8	2
Appendicitis (121)	7	3	0	3	0	0	0	1
Hernia (122[4])	7	0	0	2	0	2	1	2
Nephritis (130-131)	8	1	1	3	3	0	0	0
All Other Causes	21	3	1	5	2	0	6	4
No Secondary Cause	148	36	19	18	23	6	19	27

to this cause were charged to title 146—albuminuria and eclampsia, and 13 to title 147—other toxemias of pregnancy.

Forty-four deaths were attributed to other conditions associated with pregnancy, childbirth and the puerperal state. Of this number, 20 were charged to ectopic gestation, 18 to phlegmasia alba dolens, embolus and sudden death (not specified as septic) and 6 to other and unspecified conditions of pregnancy and the puerperal state. This group comprises only 13.8 per cent of the total mortality charged to puerperal causes during the year.

3.) *The Contributory Causes of Puerperal Deaths.* Contributory causes were reported in 172 of the 320 deaths charged to puerperal causes during 1939. Of these contributory causes, 92 were conditions of the puerperal state other than that given as the primary cause of death, while 80 were non-puerperal causes. This information is presented in Table 13.

Of the 92 deaths with puerperal secondary causes, hemorrhage was given as the contributory cause in 7 deaths; toxemia in 10 cases; cesarean section in 35, accidents of childbirth (trauma of the uterus, dystocia, abnormal presentations, etc.) in 17 and miscellaneous other puerperal conditions in 22.

Of the 80 deaths with non-puerperal secondary causes, the contributory causes were distributed as follows: non-malignant tumors of the uterus, 5; diseases of the heart and circulatory system, 14; pneumonia and influenza, 18; appendicitis, 7; hernia, 7; nephritis, 8; and other miscellaneous causes, 21.

4.) *Puerperal Deaths—Causes Confirmed by Autopsy.* Of the 320 puerperal deaths the causes of 114, or only 35.6 per cent, were confirmed by autopsy. It is obvious that postmortem examination is more necessary to determine certain causes than others. Thus, 82 per cent of the abortions, 35 per cent of the ectopic gestations, 40 per cent of the septicemias, and 27 per cent of the toxemias were verified by postmortem examination. The number of deaths charged to the various causes and the number and percentage autopsied are shown in Table 14.

5.) *Deaths from Puerperal Causes and Complications of Pregnancy.* It was mentioned above that the New York City Department of Health started to collect information regarding the complications of pregnancy in a confidential medical report on the back of birth and fetal death certificates as early as March 1938. It was, and still is, hoped that in this way

TABLE 14

CITY OF NEW YORK—
DEATHS FROM PUERPERAL CAUSES CONFIRMED BY AUTOPSY—1939

International List Number	Cause of Death	Total	Autopsied	Per cent Autopsied
140-150	Total—All Causes	320	114	35.6
140-141	Abortions (largely with sepsis)	50	41	82.0
142	Ectopic Gestation	20	7	35.0
143	Other Accidents of Pregnancy	4	2	50.0
144[1]	Placenta Praevia	16	2	12.5
144[2]	Other Hemorrhages	27	2	7.4
145	Septicemia (excluding abortions)	74	30	40.5
146-147	Albuminuria, Eclampsia and Other Toxemias	37	10	27.0
148	Puerperal Phlegmasia Alba Dolens, Embolus, Sudden Death (not septic)	18	3	16.7
149[1]	Cesarean Section	24	4	16.7
149[2-4]	Accidents of Childbirth	48	13	27.1
150	Other Puerperal Causes	2	0	0.0

valuable information might be collected to shed light on the maternal, infant and fetal mortality. It is quite obvious, from a comparison of the data given on death certificates with the analysis of the complications reported on the birth and fetal death certificates filed in connection with 235 of the 320 deaths charged to puerperal causes in 1939, that the complications are not being fully reported. Such information as has been collected on this subject is presented in Table 15.

Non-malignant tumors of the uterus were reported as the secondary cause of 5 puerperal deaths, but were not mentioned as complications of pregnancy on the birth certificates. Diseases of the circulatory system were reported as the contributory cause of 14 puerperal deaths; they were mentioned on only 12 of the birth and fetal death certificates. Similarly, although influenza and pneumonia were reported in connection with 18 puerperal deaths, these conditions were reported as complications of only 4 pregnancies. Of course, it is possible that the pneumonia developed after birth in many of these cases and was not really a complication of pregnancy.

TABLE 15

CITY OF NEW YORK — DEATHS FROM PUERPERAL CAUSES BY PRIMARY CAUSE OF DEATH — AND COMPLICATIONS OF PREGNANCY — 1939

Complication of Pregnancy	Primary Cause of Puerperal Death							
	Total—All Causes 140-150	Abortions—(largely with sepsis) 140-141	Hemorrhages 144	Septicemia (excluding abortions) 145	Toxemias 146-147	Cesareans 149[1]	Accidents of Pregnancy 149[3-4]	All other Causes 142, 143, 148, 150
Total	235	15	40	68	26	25	40	21
Syphilis (34[2])	3	0	0	1	2	0	0	0
Diseases of the Circulatory System (90-103)	12	0	2	2	2	2	3	1
Pneumonia and Influenza (11, 107-109)	4	2	0	0	0	0	2	0
Appendicitis (121)	3	1	0	2	0	0	0	0
Hernia (122[4])	1	0	0	0	0	0	1	0
Nephritis (130-131)	3	0	1	1	1	0	0	0
Puerperal Hemorrhages (144)	9	2	0	4	0	1	1	1
Toxemias of Pregnancy (146-147)	26	3	4	11	2	3	3	0
Other Complications (59, 71, 81, 106, 115, 133, 139)	9	2	0	3	2	0	1	1
No Complications	165	5	33	44	17	19	29	18

Appendicitis and hernia were each reported 7 times as contributory causes of death, but appendicitis was cited only 3 times and hernia only once as a complication of pregnancy. Nephritis was given as the secondary cause of 8 deaths whereas it was mentioned as a complication in only 3 pregnancies. Twenty-one other deaths had contributory causes mentioned on the death certificate which were not mentioned under complications of pregnancy.

On the other hand, syphilis was mentioned as a complication of pregnancy in 3 cases when it was not mentioned on the death certificate and hemorrhage was reported as a complication in 9 pregnancies but was mentioned as a contributory cause in only 7 deaths. Similarly, toxemia of pregnancy was reported as a complication of 26 pregnancies which resulted in maternal death whereas it was mentioned only 10

times as a contributory cause of death. Seven other non-puerperal conditions were reported as complications of pregnancies but were not mentioned on the death certificates. Greater care in filling out birth, fetal death and death certificates would yield more accurate information, which would, in turn, be of benefit to clinicians and obstetricians in their practices. The collaboration of physicians in making complete reports in the certificates they are required to file with the Health Department, with a view toward using this information in further efforts to reduce maternal, infant and fetal mortality, would be greatly appreciated.

Although not classified as a complication of pregnancy, it may be of interest here to state that the puerperal mortality rate associated with multiple births was 52.3, whereas the general rate was 29.6.

IX. Operative Procedures Associated with Puerperal Deaths

Operative procedures were reported in connection with 147 of the 235 puerperal deaths for which supplementary data were available from the confidential reports made on birth and fetal death certificates. Principal among these were:

Cesarean section	68
Low forceps (only)	23
Version and extraction	14
Episiotomy (with or without forceps)	10
Mid and high forceps (only)	9

Because of the great interest in the subject, particular attention has been given to cesarean section.

1.) *Delivery by Cesarean Section.* Of the 108,018 reported terminated pregnancies, 2,558 were terminated by cesarean operation. This number is 2.4 per cent of the total. The same rate holds for the white women, while among the colored it was 1.8 per cent. Except for home deliveries, the performance of the cesarean section was least practiced in the municipal hospitals where only 1.5 per cent of the white and 1.8 per cent of the colored deliveries were made by this method. In the proprietary hospitals, where the patients are predominantly white, cesarean sections were done in 1.9 per cent of the deliveries. In the voluntary hospitals, cesarean section was employed in slightly more than 3 per cent of all births. These facts are shown in Table 16.

2.) *Mortality Associated with the Cesarean Operation.* Of the 2,558

TABLE 16

CITY OF NEW YORK — PREGNANCIES TERMINATED BY CESAREAN OPERATIONS RATE PER 100 REPORTED TERMINATED PREGNANCIES BY COLOR AND TYPE OF INSTITUTION — 1939

Type of Institution	Number*	Total Reported Terminated Pregnancies			Pregnancies Terminated by Cesarean					
					Number			Per Cent		
		T	W	C	T	W	C	T	W	C
New York City—Total		108,018	99,559	8,459	2,558	2,405	153	2.4	2.4	1.8
Municipal	17	18,639	13,470	5,169	292	201	91	1.6	1.5	1.8
Voluntary—City Aid	57	50,278	48,169	2,109	1,543	1,486	57	3.1	3.1	2.7
Voluntary—No City Aid	21	9,463	9,443	20	316	316	0	3.3	3.3	—
Proprietary	63	21,318	21,193	125	400	395	5	1.9	1.9	**
Other Government Institutions	7	231	228	3	4	4	0	1.7	1.8	—
Home		7,952	6,953	999	3	3	0	0.4	0.4	—
Unknown		137	103	34	0	0	0	—	—	—

*Number of hospitals in which at least 1 pregnancy terminated.
**Numbers too small to yield a statistically significant rate.

women who were delivered by cesarean section, 68 died. Of these 68 deaths, 43 were charged to other causes than cesarean (including 10 to hemorrhage, 20 to septicemia and 4 to toxemias of pregnancy) and 35 to cesarean—no other puerperal cause being given on the certificate. These women who died constitute 2.7 per cent of the women who were delivered by cesarean (2.5 per cent of the white, 5.9 per cent of the colored). In this connection, it is of interest that 36 of the 68 women who died after cesarean section went into labor before the operation was performed. Of the remaining 32, 10 were reported to have had no labor while the question was unanswered in 22 cases.

The percentage of deaths was highest in the municipal hospitals, where 5.1 per cent of the cesarean sections were followed by death—4.5 per cent among white women and 6.6 per cent among colored women. Obviously, the character of the clientele of the municipal hospitals must be borne in mind when considering these figures. In the voluntary hospitals, approximately 2.0 per cent of the cesarean sections

TABLE 17

CITY OF NEW YORK—MORTALITY ASSOCIATED WITH CESAREAN OPERATION FATALITY RATE PER 100 OPERATIONS BY COLOR AND TYPE OF INSTITUTION—1939

Type of Institution	Number*	Total Cesareans			Deaths Following Cesareans					
					Number			Per Cent		
		T	W	C	T	W	C	T	W	C
New York City—Total		2558	2405	153	68	59	9	2.7	2.5	5.9
Municipal Hospitals	15	292	201	91	15	9	6	5.1	4.5	6.6
Voluntary—City Aid	47	1543	1486	57	34	32	2	2.2	2.2	3.5
Voluntary—No City Aid	18	316	316	0	6	6	0	1.9	1.9	—
Proprietary Hospitals	44	400	395	5	13	12	1	3.3	3.0	**
Other Government Institutions	4	4	4	0	0	0	0	—	—	—
Home		3	3	0	0	0	0	—	—	—

*Number of hospitals in which at least 1 Cesarean was performed.
**Numbers too small to yield a statistically significant rate.

resulted fatally; in the proprietary hospitals, 3.3 per cent. Table 17 shows these facts.

We had hoped to be able to report on the relative fatality rates for the various types of cesarean operations now in use, but unfortunately, in spite of a request for specific information, the responses to item 30 on the birth and fetal death certificates have generally failed to describe the type of cesarean operation performed and it has consequently been impossible for us to develop this interesting information.

3.) *Relation of Cesarean Mortality to Total Puerperal Mortality.* Although it has been shown that only 2.4 per cent of all the reported pregnancies were terminated by cesarean section and that 2.7 per cent of the cesarean sections were followed by the death of the mother, the role of this operation in the puerperal death rate is revealed only when the number of deaths following cesareans is referred to the total of puerperal deaths. When this is done, we see that of all puerperal deaths, 21.3 per cent were associated with cesarean operation. In the municipal hospitals, the ratio was 12.6 per cent; in the voluntary hospitals,

TABLE 18

CITY OF NEW YORK — MORTALITY ASSOCIATED WITH CESAREAN OPERATION RATE PER 100 PUERPERAL DEATHS BY COLOR AND TYPE OF INSTITUTION — 1939

Type of Institution	Number*	Total Puerperal Deaths			Deaths Following Cesareans					
					Number			Per Cent		
		T	W	C	T	W	C	T	W	C
New York City—Total		320	256	64	68	59	9	21.3	23.0	14.1
Municipal Hospitals	17	119	67	52	15	9	6	12.6	13.4	11.5
Voluntary—City Aid	57	127	119	8	34	32	2	26.8	26.9	25.0
Voluntary—No City Aid	21	24	24	0	6	6	0	25.0	25.0	—
Proprietary	63	38	37	1	13	12	1	34.2	32.4	**
Other Government Institutions	7	0	0	0	0	0	0	0.0	0.0	—
Home	—	12	9	3	0	0	0	0.0	0.0	—

*Number of institutions in which at least one pregnancy terminated.
**Numbers too small to yield a statistically significant rate.

approximately 26 per cent and in the proprietary hospitals, 34.2 per cent. These facts are presented in Table 18.

In considering these percentages, it must be borne in mind that the puerperal death rates from all causes, by type of institution, are in exactly the reverse order. The mortality from cesarean section in proprietary hospitals is relatively high; the total puerperal death rate in these institutions, as was shown in Table 9, is relatively low.

It must also be realized, that as a general rule cesarean section is practiced only because birth by the normal process is deemed hazardous to the life of the mother or the infant, or both. Although cesarean section is chosen as the method of delivery in many cases as a result of careful prenatal study, in many other instances it is employed in a last desperate effort to save the life of the mother or the infant, when the saving of that life appears impossible if labor is permitted to continue. Under such circumstances, it is not surprising that the puerperal mortality following cesarean section is relatively high.

TABLE 19

CITY OF NEW YORK — PUERPERAL AND MATERNAL MORTALITY — 1939

Puerperal Mortality Rate				Maternal Mortality Rate			
	Total	White	Colored		Total	White	Colored
Reported Terminated Pregnancies — Total	108018	99559	8459	Reported Terminated Late Pregnancies — Total	104684	96551	8133
Single Live Births	100190	92552	7638	Single Live Births	100190	92552	7638
Single Fetal Deaths — Total	6594	5905	689	Single Fetal Deaths (over 20 weeks gestation)	3355	2959	396
Multiple Births — Total	1149	1048	101	Multiple Births (over 20 weeks gestation)	1109	1019	90
Undelivered women dying from Puerperal Causes (140-150) — Total	85	54	31	Undelivered women dying from Late Puerperal Causes (144-150)	30	21	9
Deaths from Puerperal Causes — (140-150)	320	256	64	Deaths from Late Puerperal Causes (144-150)	246	205	41
Puerperal Death Rate	29.6	25.7	75.7	Maternal Death Rate	23.5	21.2	50.4

X. MATERNAL MORTALITY

During the course of the preceding discussion, we have considered the total number of deaths resulting primarily from puerperal causes, early or late, and we have based all of our rates on the total terminated pregnancies for which we have reports. Before we close, we should like to discuss briefly *maternal* mortality as compared with *puerperal* mortality. At the beginning of this paper, we defined a maternal mortality rate as the number of deaths resulting primarily from a puerperal cause associated with late pregnancy, per ten thousand reported terminated late pregnancies. The base includes all live single births, fetal deaths of 20 weeks gestation and over, multiple births of 20 weeks gestation and over, and undelivered women dying of causes associated with late pregnancy. The *maternal* mortality rate is calculated for the purpose of representing mortality associated with child-bearing as contrasted with the *puerperal* mortality rate which includes mortality associated with pregnancy in all its stages—abortion and ectopic gestation,

as well as that associated with childbirth at or near term. Data used in this calculation are presented in Table 19.

This table presents the data included in the calculation of each of these rates, by color. In deriving the maternal mortality rate, we use as a base 104,684, which represents the number of women exposed to the risk of dying from puerperal causes associated with late pregnancy in New York City during 1939. Of this number, 246 died. This yields a maternal mortality rate of 23.5 per 10,000 reported terminated late pregnancies, a figure considerably lower than the puerperal mortality rate—29.6 per 10,000 total reported terminated pregnancies. For white women, the maternal mortality rate was 21.2, and the puerperal mortality rate, 25.7. The maternal mortality rate for colored women was 50.4 as compared with a puerperal mortality rate of 75.7. The rates for the colored are much more affected by this difference in method of computing mortality rates. However, it should be noted that the figures involved in the colored group are very much smaller than those for the white group; consequently any change in grouping would affect the rates for the colored much more seriously than for the white, although such changes might not actually reflect a significant difference. In the case under discussion, even discounting for the sensitivity of small numbers and the statistical inadequacy of the sample, there is an interesting difference between the behavior of rates for the white and colored. Of the 64 puerperal deaths reported for the colored group, 41, or 64.1 per cent, were associated with late pregnancy, while for the white group, 205, or 80.1 per cent, of the 256 puerperal deaths, were connected with late pregnancy. This observation leads to the conclusion that proportionately more puerperal deaths among the colored occur in early pregnancy than among the whites. It has already been shown that the mortality attributed to abortions is much higher among colored than among white women.

Knowing that the *maternal* death rate among the colored is approximately two and one-half times that of white women, it is manifestly unfair to compare the "crude" rates for the several boroughs, because of the differences in their color composition. For example, Manhattan reported only three-fourths as many terminated pregnancies among white women, but almost twice as many among the colored, as did Brooklyn. Manhattan reported the lowest maternal mortality among white women, Brooklyn next and Richmond third.

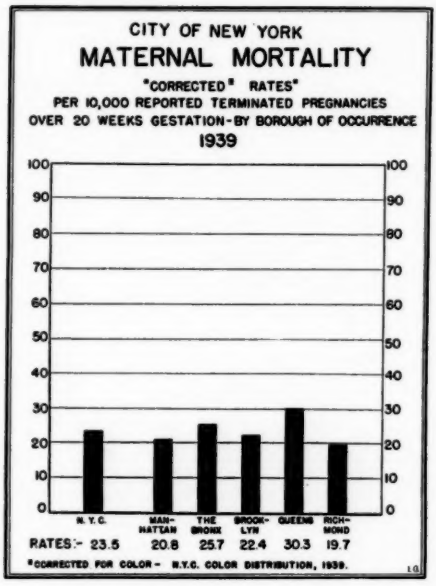

Chart VII

TABLE 20

CITY OF NEW YORK — MATERNAL DEATH RATES — BY BOROUGHS — BY COLOR — RATES PER 10,000 LATE PREGNANCIES TERMINATED — 1939

Borough	White	Colored	Death Rates per 10,000	
			Crude	Corrected*
Entire City	21.2	50.3	23.5	23.5
Manhattan	18.0	53.8	22.9	20.8
Bronx	26.4	**	26.1	25.7
Brooklyn	20.1	49.7	21.8	22.4
Queens	26.8	**	28.4	30.3
Richmond	21.4	0.0	20.7	19.7

*Corrected for differences in color distribution.
**Numbers too small to yield statistically significant rates.

If we correct the *maternal* mortality rates to eliminate the effects of differences in the colored population in the various boroughs, we obtain results that are presented in Table 20 and in Chart VII.

It is to be noted that the *maternal* mortality rate does not include deaths associated with abortions and ectopic pregnancies. When these rates for the several boroughs are corrected for color differences, Richmond has the lowest rate, Manhattan is second, Brooklyn third, Bronx fourth and Queens highest.

Conclusion

This paper has aimed at presenting certain salient facts about puerperal mortality in New York City. We are aware of the fact that it does not cover every point that might interest this group, but time does not permit a more extensive treatment of the subject on this occasion. More material is available on this sample of 320 puerperal deaths. But even more important is the fact that we have established a base from which comparisons may be made in the future. It should be borne in mind that one year's experience can serve only as a springboard for further efforts. At the close of 1940, we shall be able to give you a similar analysis for a two-year period, which will include approximately twice as many cases. Perhaps more decided tendencies will be exhibited in this greater body of material; certainly, in many cases we shall be able to establish statistical validity where we have been hampered by a small sample this year.

I must point out, however, that any report that I can make to you cannot rise any higher than the information you report on the certificates you fill out. Your coöperation in giving complete responses to the questions will be of mutual assistance.

It has been a pleasure and a privilege to present this information to you. I hope that at some future time, I shall have the opportunity of reporting to you the results of further investigation.

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DEATHS OF FELLOWS

JACOBY, GEORGE WASHINGTON: 44 West 72 Street, New York City; born in St. Louis, Missouri, September 21, 1856; died in New York City, September 11, 1940; graduated in medicine from the Bellevue Hospital Medical College of New York City in 1877, and from the University of Berlin, Germany, in 1878; elected a Fellow of the Academy June 4, 1885.

At the time of his death, Dr. Jacoby was consulting neurologist to the Lenox Hill and Beth Israel Hospitals and to the Craig Colony for Epileptics. From 1882 to 1902 he was attending physician to the German Dispensary of New York. At the Women's Medical College of the New York Infirmary, he was lecturer on nervous diseases, 1887—1889; clinical professor of nervous diseases, 1890—1895; and professor of nervous and mental diseases, 1895—1900.

Dr. Jacoby was a Fellow of the American Medical Association, a member of the New York Neurological Society and its president, 1888-90, a member of the American Neurological Association and its president in 1915, and a member of the State and County Medical Societies. He was the author of numerous books and medical monographs on subjects related to his specialty.

KNOPF, SIGARD ADOLPHUS: 16 West 95th Street, New York City; born in Halle-on-the-Saale, Germany, November 27, 1857; died in New York City, July 15, 1940; graduated in medicine from Bellevue Hospital Medical College of New York City in 1888; received the degree of A. B. from the Sorborne, Paris, in 1890 and graduated from the medical school of the University of Paris in 1895; elected a Fellow of the Academy, March 4, 1897.

Dr. Knopf was professor of medicine in the department of physiotherapy at the New York Post-Graduate Medical School, Columbia University, 1908-1920, and senior visiting physician to the Health Department's Riverside Tuberculosis Hospital, 1906-1922. He was one of the founders of the New York and the National Tuberculosis Association, an honorary member of the American Association for Thoracic Surgery and the American Tuberculosis Association, and a

member of the Association of Military Surgeons of the United States, the Society of Medical Jurisprudence, the American Heart Association, the American Society for Physical Research, the American Association for the Advancement of Science and the County and State Medical Societies.

Dr. Knopf was the recipient of many honors, among which were the Alvaranga Prize of the College of Physicians of Philadelphia for his treatise on pulmonary tuberculosis, 1898; the prize of 4,000 marks given in 1899 by the International Tuberculosis Association at Berlin for his essay entitled: "Tuberculosis as a Disease of the Masses and How to Combat It," which has been translated into thirty languages. In 1932, he represented this country at the Congress of the International Union against Tuberculosis held at The Hague.

In the World War, Dr. Knopf was a captain in the Medical Reserve Corps and at his death held the rank of major in the Officers Reserve.

Dr. Knopf was the author of over four hundred books and pamphlets on medical and related subjects.

LEVENE, PHOEBUS AARON THEODORE: 129 East 82 Street, New York City; born in Sagar, Russia, February 25, 1869; died in New York City, September 8, 1940; graduated in medicine from the Imperial Military Medical Academy of St. Petersburg, Russia in 1891; elected a Fellow of the Academy January 7, 1932.

Dr. Levene was member emeritus of the Rockefeller Institute for Medical Research, joining that institution as an assistant in chemistry in 1905 and being elected to membership in 1907. Dr. Levene came to this country in 1892 and began the practice of medicine in New York City, meanwhile enrolling as a special student in chemistry at Columbia University. He studied also in Berne, Switzerland, and in Berlin, Marburg and Munich, Germany. In 1896 he became an associate in chemistry for research at the State Pathological Institute of New York. With the exception of 1900 to 1902, which he spent at the Saranac Laboratory for the Study of Tuberculosis, he held the post at the Institute until 1905 when he joined the staff of the Rockefeller Institute. He was Herter lecturer in pathological chemistry at New York University 1905 to 1906.

The Willard Gibbs Medal of the Chicago section of the American Chemical Society was awarded to Dr. Levene in 1931, with a citation which described him as "the outstanding American worker in the application of organic chemistry to biological problems" and which mentioned especially his work in stereochemistry and his success in concentrating Vitamin B-2. In 1938, the William H. Nichols medal came to him from the New York section of the American Chemical Society for his "study of the configurational relationships of the simpler optically active organic compounds."

Dr. Levene was a member of the Bavarian Academy, the Royal Society of Upsala, Sweden, the Royal Society of Medical and Natural Science of Brussels and other German, French and Swiss scientific societies. He was a member of the National Academy of Sciences, the American Philosophical Society, the American Chemical Society, the American Physiological Society, the Harvey Society, the Society of Biological Chemists, the Society of Experimental Biology and Medicine, and a Fellow of the American Association for the Advancement of Science.

Dr. Levene was the author of numerous papers on proteins, nucleins, carbohydrates, lipoids, problems of stereochemistry, and of the monographs, "Hexosamines and Mucoproteins," 1925, and "Nucleic Acids," 1931.

VAN DER BOGERT, FRANK: 111 Union Street, Schenectady, New York; born in Schenectady, October 4, 1873; died in Schenectady, September 24, 1940; graduated in medicine from the University of Pennsylvania School of Medicine in 1900; elected a Fellow of the Academy February 3, 1916.

Dr. van der Bogert was senior pediatrician to the Ellis Hospital; special lecturer in pediatrics at the Albany Medical College; Chairman of the Medical Advisory Committee, Schenectady Public Schools; a diplomate of the American Board of Pediatrics; a Fellow of the American College of Physicians, the American Academy of Pediatrics, the American Medical Association, and a member of the State and County Medical Societies.